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EXPERIENCES WITH A NEW ANTICOAGULANT*

GEORGE W. ALLEN, M. D.

AND

EDGAR HULL, M. D.

NEW ORLEANS

When dicumarol, the active agent of spoiled sweet clover disease in cattle, was first isolated by Campbell and Link[1] in 1940, an extensive investigation was launched to determine its possible therapeutic value in conditions of intravascular clotting. As statistical studies progressed and methods of control improved, wide acceptance was established in thromboembolic disease. However, it was also proven that there are definite limitations to its use. It is necessary to control the drug with a rather tricky laboratory procedure, the prothrombin time. There is on occasion a definite hemorrhage tendency with dicumarol which may or may not be anticipated by the level of prothrombin deficiency. In addition it has been shown that dicumarol can produce injurious lesions of small blood vessels which may cause a hemorrhagic diathesis.[2] This diapodesis of red cells through vessel walls not infrequently causes alarming or even occasional fatal uncontrollable hemorrhage.[3] It is this latter type of toxicity which the individual using dicumarol has most to fear.

As these limitations came to light, research was continued with agents of a similar chemical structure.[4] The most promising of these to date is 3, 4-(2′-methyl-2′-methoxy-4′-phenyl) - dihydropyranocoumarin, * which bears Link's code number of sixty-three.

Battle *et alii*[5] have indicated from using compound 63 in dogs, that prothrombin levels may be kept at much smaller concentrations without incurring hemorrhage than can be done with dicumarol. They also noted that the small blood vessels appeared uninjured even after massive doses of compound 63. In addition, clinical trials were undertaken in humans. Weight for weight, compound 63 appeared to be two to three times as potent in depressing the prothrombin as was dicumarol.

The purpose of this study was to further the clinical observations and to evaluate the effect upon the coagulation time.

METHODS

Prothrombin times were done using Difco thromboplastin by a modification of the Quick[6] method in which the calcium and thromboplastin were premixed. Standardization and dilution curves were done as suggested by Hurn *et alii*.[7] Coagulation times were done by a modification of the Lee-White technic which gives normals of twenty-five to thirty-five minutes.

Presented at the Seventieth Annual Meeting of the Louisiana State Medical Society, April 26, 1950, in Baton Rouge.

*From the Department of Medicine of the Louisiana State University School of Medicine, and the Charity Hospital of Louisiana at New Orleans.

*BL-5 Abbott Laboratories. We wish to thank the Abbott Laboratories for making a supply of the drug available for investigation.

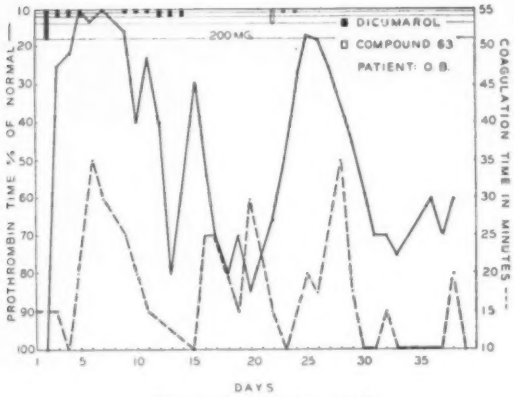

Figure 1. Patient O.B.

Patients entering the hospital with thromboembolic disease were treated with either compound 63 or both compound 63 and dicumarol. Sixteen patients are included in the series with 8 illustrated in some detail.

RESULTS

The first figure pertains to a patient who was dicumarolized and maintained for several days. His therapy was then allowed to lapse. After a rest interval he was given doses of compound 63 which in milligrams equaled a little less than half that of dicumarol. In this instance the response was not as rapid nor as great. The effects upon the coagulation time were similar. Figure 2 represents a similar procedure. A maintenance dose of compound 63 one-quarter that of dicumarol was not quite as effective. The coagulation times were again similar.

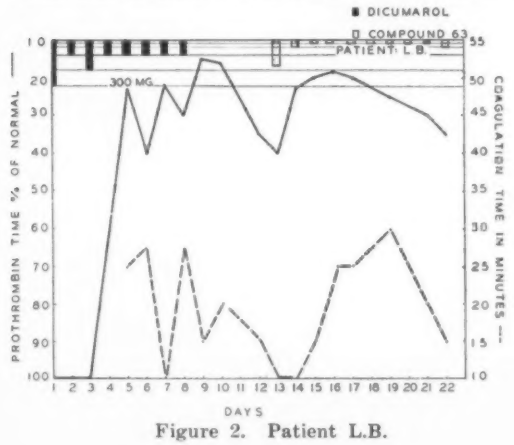

Figure 2. Patient L.B.

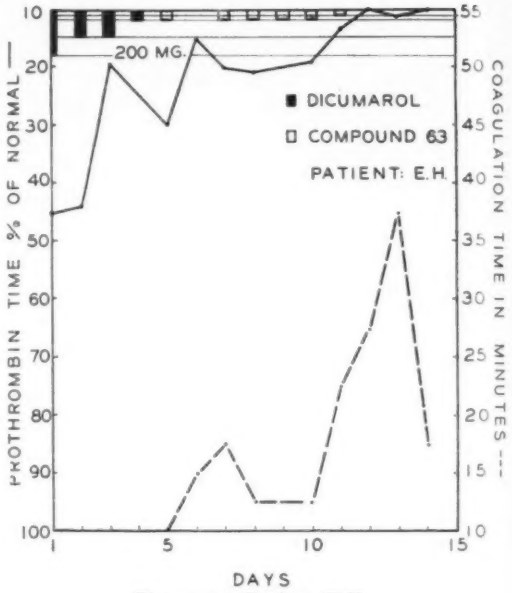

Figure 3. Patient E.H.

Figure 3 is of a patient first dicumarolized, and then maintained on compound 63 with approximately one-half the anticipated dose of dicumarol. The increasing coagulation time here is thought due to the diminishing prothrombin rather than to a particular of compound 63. These results would confirm the opinion of Battle *et alii*[5] that compound 63 has two to three times the potency of

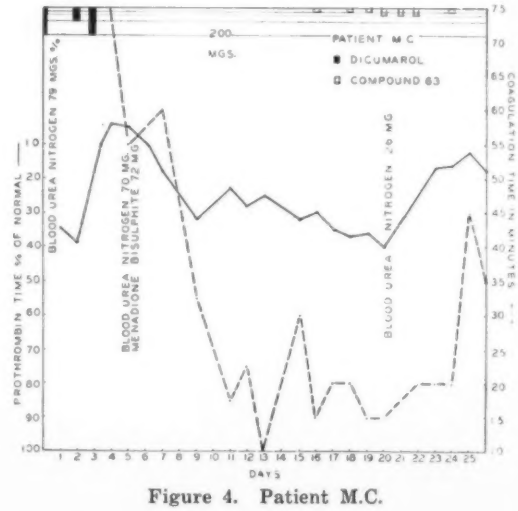

Figure 4. Patient M.C.

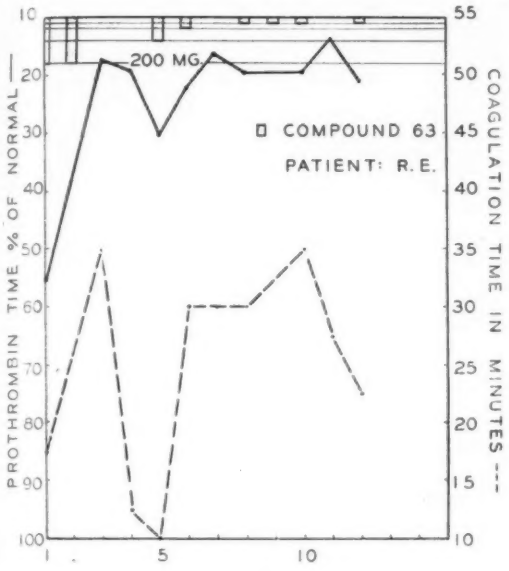

Figure 5. Patient R.E.

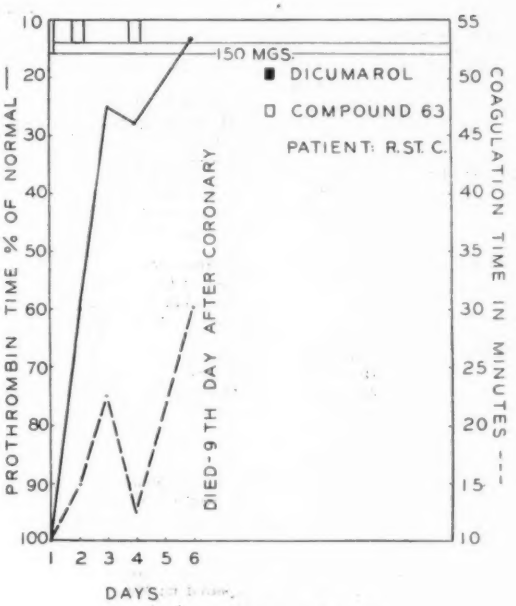

Figure 7. Patient R.St.C.

dicumarol. Dosages used in other patients were governed accordingly. Figure 4 is of a patient with a myocardial infarct, shock, and uremia, who hyperacted to dicumarol and hence was given menadione bisulphite.

The marked effect upon the coagulation time is noted. Later she was cautiously given compound 63. When larger doses were necessary than expected, note was again taken of renal function and it was

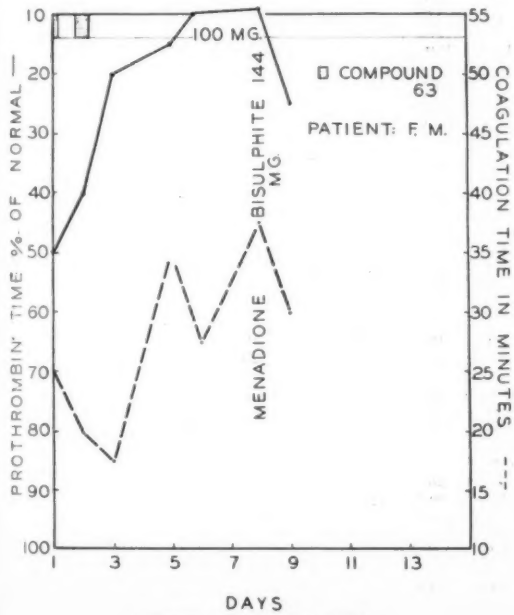

Figure 6. Patient F.M.

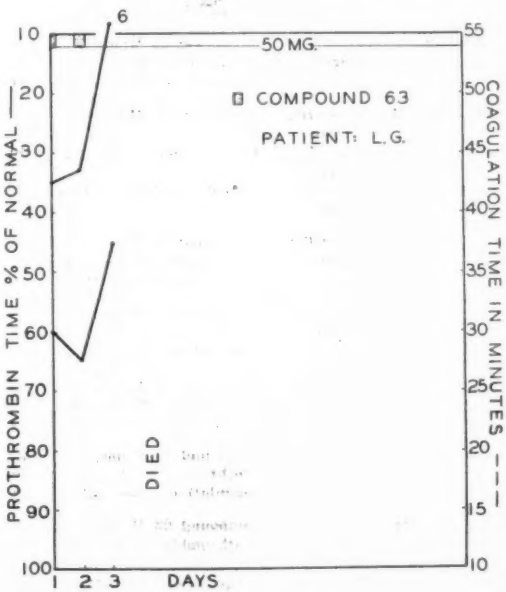

Figure 8. Patient L.G.

TABLE 1

No.	Pt.	Age	Sex	Diagnosis	Days	1	2	3	4	5	6	7	8	9	10
1	EH	67	M	Cong. H. F Pulmonary Infarct	Dicumarol—Mgms.	200	100	100	50	.....	.....	.....	.....	...	.....
					Compound 63 Mgms.	.....	.....	.....	.....	50	.....	50	50	50	50
					Prothrombin % Normal	45	44	20	.....	30	15	20	21	....	19
					Coagulation Time—Mins.	.....	.....	.....	.....	10	15	17.5	12.5	....	12.5
2	MC	67	F	Myocardial Infarct Uremia	Dicumarol—Mgms.	200	100	200	.....	.....	.....	.....	.....	....	.....
					Compound 63 Mgms.	.....	.....	.....	.....	.....	.....	.....	.....	...	.....
					Prothrombin % Normal	.....	34.5	39	.....	4	6	.....	18	....	32
					Coagulation Time—Mins.	.....	.....	.....	.....	75	55	.....	60	....	32.5
3	OB	50	M	Myocardial Infarct	Dicumarol—Mgms.	.....	200	50	50	25	.....	.....	.....	25	25
					Compound 63 Mgms.	.....	.....	.....	.....	.....	.....	.....	.....	...	.....
					Prothrombin % Normal	100	100	25	22	10	13	10	.....	16	40
					Coagulation Time—Mins.	15	15	15	10	20	35	30	.....	25	20
4	JM	33	M	Rheumatic Heart Dis. Auricular Fibrillation with Embolism	Dicumarol—Mgms.	25	50	25	50	25	50	25	50	25	50
					Compound 63 Mgms.	.....	.....	.....	.....	.....	.....	.....	.....	...	.....
					Prothrombin % Normal	30	.....	33	.....	29	.....	. .....	21	...	.....
					Coagulation Time—Mins.	45	.....	45	.....	52.5	.....	.....	47.5	...	.....
5	LB	62	M	Myocardial Infarct	Dicumarol—Mgms.	300	100	200	100	100	100	100	100	....	.....
					Compound 63 Mgms.	.....	.....	.....	.....	.....	.....	.....	.....	....	.....
					Prothrombin % Normal	100	100	100	.....	23	40	22	30	15	16
					Coagulation Time—Mins.	.....	.....	.....	.....	25	27.5	10	27.5	15	20
6	DP	67	M	Myocardial Infarct	Dicumarol—Mgms.	.....	.....	.....	.....	.....	.....	.....	.....	....	200
					Compound 63 Mgms.	100	75	.....	.....	.....	.....	.....	.....	....	.....
					Prothrombin % Normal	100	.....	8	20	20	22	25	33	....	33
					Coagulation Time—Mins.	.....	.....	40	32.5	.....	27.5	22.5	10	....	22.5
7	JR	67	M	Myocardial Infarct	Dicumarol—Mgms.										
					Compound 63 Mgms.	.....	100	.....	.....	.....	.....	.....	.....	....	.....
					Prothrombin % Normal	38	.....	36	22	18	15	.....	22	....	32
					Coagulation Time—Mins.	15	25	20	15	15	35	.....	.....	....	25
8	RE	72	M	Myocardial Infarct	Compound 63 Mgms.	150	150	.....	.....	100	50	.....	25	25	25
					Prothrombin % Normal	55	.....	17	19	30	22	16	19.5	....	19.5
					Coagulation Time—Mins.	17.5	.....	35	12.5	10	30	30	30	...	35
9	FM	90	M	Anginal Attack	Compound 63 Mgms.	100	100	.....	.....	.....	.....	.....	.....	...	.....
					Prothrombin % Normal	50	40	20	.....	15	10	.....	8.5	15	.....
					Coagulation Time—Mins.	25	20	17.5	.....	35	27.5	.....	37.5	30	.....
10	WG	39	M	Myocardial Infarct	Compound 63 Mgms.	100	100	100	50	.....	50	50	25	25	25
					Prothrombin % Normal	45	.....	35	17	12	13.5	19	12	....	14
					Coagulation Time—Mins.	37.5	.....	45	10	47.5	.....	30	32.5	....	20
11	EJ	47	M	Luetic Ht. Disease Cong. Ht. Failure Anginal Attack	Compound 63 Mgms.	50	50	50	50	.....	.....	.....	.....	....	.....
					Prothrombin % Normal	40	35	35	.....	16	.....	40	35	....	.....
					Coagulation Time—Mins.	10	12.5	15	.....	30	.....	20	20	....	.....
12	JS	69	M	Myocardial Infarct	Compound 63 Mgms.	100	100	50	.....	.....	.....	.....	.....	....	.....
					Prothrombin % Normal	50	50	30	12	10	13	38	.....	42	.....
					Coagulation Time—Mins.	10	12.5	30	20	35	30	22.5	.....	20	.....
13	NL	75	M	Myocardial Infarct	Compound 63 Mgms.	100	100	.....	.....	.....	.....	50	.....	....	.....
					Prothrombin % Normal	40	35	10	8.5	.....	10	8.5	12	....	.....
					Coagulation Time—Mins.	17.5	17.5	12.5	.....	.....	20	25	15	....	.....
14	TH	67	M	Myocardial Infarct	Compound 63 Mgms.	100	50	50	50	.....	.....	.....	.....	....	.....
					Prothrombin % Normal	100	50	27	.....	8	.....	19	35	30	28
					Coagulation Time—Mins.	10	10	10	10	10	.....	10	10	10	10
15	R St C	72	M	Myocardial Infarct	Compound 63 Mgms.	150	100	.....	100	.....	.....	.....	.....	....	.....
					Prothrombin % Normal	100	60	25	28	.....	13	.....	.....	....	.....
					Coagulation Time—Mins.	10	15	22.5	12.5	.....	30	.....	.....	....	.....
16	LG	72	M	Myocardial Infarct Diabetes Mellitus. Pulmonary Fibrosis	Compound 63 Mgms.	50	50	.....	.....	.....	.....	.....	.....	....	.....
					Prothrombin % Normal	35	33	6	.....	.....	.....	.....	.....	....	.....
					Coagulation Time—Mins.	30	27.5	37.5	.....	.....	.....	.....	.....	....	.....

TABLE 1

11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32
25																					
13.5	10	11	10																		
22.5	27.5	37.5	17.5																		
						25		25	25	50	50	50		25							
	22	28	25		32	30	35	37	36	40		24	17	16	12	17.5					
	17.5	22.5	10		30	15	20	20	15	15		20	20	20	45	35					
25	50	50	50								100	25	25								
23	40	80		30	55	70	80	70	85		65	50	27	17	18	25		45		70	70
15				10	25	25	20	15	30		15	10	10	20	12.5	35		17.5	10	10	15
25	50	25	50	25	50	25	50	25	50	25	50	25									25
													25	25	25	25	25	25			
	50			55				30			50		50		40		17		11		16
	50			40				32.5			32.5		42.5		20		27.5		52.5		60
		150	50	25	25	25	25	25	25	25	50										
	35	40	23	20	19	20		25		30	35										
	15	10	10	15	25	25		30		20	15										
150																					
		50				50															
30	15	25	16	17		22	13.5	12													
	27.5	20	30			12.5	32.5	30													
		100					100	50	50												
38	40	40		28	26	30	37	30	25												
10	10	10		10	10	10	15	10	12.5												
	25																				
13.5	21																				
27.5	22.5																				
	25																				
10	16																				
30	25																				

found to have markedly improved. It has been our observation that there is more importance to be attached to the ability of the kidney to excrete the anticoagulant than there is to original prothrombin depression. Figure 5 is a typical example of patients being maintained on compound 63. Figure 6 is of a patient 90 years of age who was given menadione bisulphite because of continuing prothrombin depression. There was a prompt reversal of the effect within twelve hours. Figures 7 and 8 are the two deaths which have occurred. Unfortunately post mortems were obtained in neither. Patient R. St. C. was clinically progressing well, but on the ninth day of his illness abruptly sat up in bed and died. Patient L. G. was seriously ill upon admission. His progress was downhill until the day of his death. An observation a few minutes before death revealed a very rapid irregular heart rate. The patient's death occurred shortly after the blood specimen was drawn for prothrombin determination. Had he lived he would have been given vitamin K. It was the clinical opinion that both these deaths were due to ventricular fibrillation. There was no detected occurrence of thromboembolic complication in any of the patients. The only hemorrhagic incident was the development of ecchymotic areas in patient F. M. around the site of, and thought due to repeated venipuncture. Table 1 is a composite of all patients included in this report.

DISCUSSION

The work of Battle *et alii* is confirmed in that the relative potency of compound 63 is two to three times that of dicumarol. It would be impossible to say whether its effect or excretion is more rapid than dicumarol in this series of cases, though there were no hemorrhagic incidents other than that described. It is also impossible to say that compound 63 may safely be used to prolong prothrombin times beyond those customarily employed. Compound 63 is thought to influence the coagulation time as does dicumarol. The response to vitamin K in one patient receiving compound 63 was similar to that expected in patients receiving dicumarol. The work with animals, which showed an absence of the toxic lesions of small blood vessels, should encourage wide adoption of the preparation as soon as current clinical studies are complete. Due to its function of depressing the formation of prothrombin it is felt that the use of the drug should be controlled by prothrombin determinations.

SUMMARY

1. The use of compound 63 in 16 patients is reported.

2. The relative potency is thought to be two or three times that of dicumarol.

3. The effects of compound 63 and dicumarol upon the coagulation time are similar.

4. The only hemorrhagic incident was an ecchymosis at the site of repeated venipuncture.

5. There were two deaths which were clinically attributed to ventricular fibrillation.

6. One patient on compound 63 was given vitamin K. A good response occurred.

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DISCUSSION

H. Guy Riché, Sr. (Baton Rouge): I have nothing to add to the author's paper but would like to offer myself to those present as an example of one who has taken dicumarol.

Within the past year I suffered several minute

retinal hemorrhages in my left eye, and was advised to have a thorough physical examination which I did at the Ochsner Clinic in New Orleans where it was found that I had a blood pressure above normal limits and some impairment of vision.

Twelve years ago, I lost my right eye following a massive retinal hemorrhage. Following this last episode, I entered Our Lady of the Lake Sanitarium where I was given dicumarol in fairly large doses for several weeks. Blood tests were made every morning. I regained the vision in my left eye and the only discomfort suffered following dicumarol was a gastrointestinal disturbance.

Dr. M. W. Hunter (Monroe): What is your impression of dicumarol dissolving a clot which was already formed?

Dr. Allen: There is probably very little effect. The great value of the drug is the prevention of extension or recurrence of the clot.

Dr. H. M. Horack (New Orleans): Did the administration of Vitamin K provoke a more rapid response in the patient on compound 63 than would have been seen with dicumarol?

Dr. Allen: For the dosage used this was a fairly typical response.

Dr. H. Guy Riché, Sr.: Do you think heparin is coming back?

Dr. Allen: Heparin has a definite place but, due to the short duration of action, the difficulty in maintaining a constant effect, and its expense in this country, it has not been as widely used as dicumarol. As far as the therapeutic value is concerned there are those who consider it superior to dicumarol. In acute conditions in which immediate responses are desired, heparin is the drug of choice.

TICK PARALYSIS: IMPLICATING *AMBLYOMMA MACULATUM**

RALPH S. PAFFENBARGER, JR., M. D.

THOMASVILLE, GEORGIA

Arthropods of medical importance include the ixodid ticks, which are vectors of such familiar diseases as spotted fever and tularemia. Certain species of Ixodidae have been incriminated in a less well-known syndrome, commonly referred to as tick paralysis. This disease occurs in both man and domestic animals. It results from a tick bite, and is characterized by ascending, flaccid paresis which occasionally may terminate in death. Removal of the tick, however, usually is followed by complete and rapid recovery. In this country, tick paralysis has been reported sporadically from widely separated areas. It is described most commonly in areas heavily infested with ticks of the genus *Dermacentor* and probably occurs more frequently than is realized. Inhabitants of endemic areas knowingly search for ticks, if certain neurological symptoms appear.[1]

This paper presents the essential features of tick paralysis and 2 case reports. One case was associated with *Amblyomma maculatum*, a tick not previously implicated in this syndrome. Extensive reviews of the subject have been written by Stanbury and Huyck[2] and by Abbott.[3] The reader is referred to these for more comprehensive clinical and experimental data.

The etiology of tick paralysis remains obscure. No infectious agent has been isolated. The disease is believed to result from the inoculation of some unidentified, tick-generated, toxic substance which appears to be neurotropic. Neither the origin of such a substance nor its method of transmission is understood.

Disease apparently results from attachment of the tick to any of several tegumental regions. The scalp, back, and groin are the sites of implantation most frequently described. Attachment in obscure areas may delay detection for sufficient time that a toxic substance could be produced.[4-6] Female ticks, many of which have been reported as gravid, are associated most frequently with tick paralysis. The fact that male ticks usually feed for only short periods may explain the association of this disease with the longer-feeding females.

Pathological changes are not demonstrated always. Those which have been described appear to be limited to the brain and spinal cord. Diffuse hyperemia with focal agranulocyte infiltration may exist. Small hemorrhagic foci have been seen within the spinal cord. Animal experiments have shown myoneural junctions and peripheral nerves to be unaffected. It is

*From the Department of Pediatrics, Louisiana State University School of Medicine, the Charity Hospital of Louisiana at New Orleans, and the Communicable Disease Center, Public Health Service, Federal Security Agency, Atlanta.

probable that a toxic substance exerts its action upon the anterior horn cells of the spinal cord and in the cells of the cranial nerve nuclei.

Onset of symptoms is frequently sudden with rapid progression. Malaise, cephalalgia, irritability, and lassitude may exist a few hours before the onset of ascending flaccid paralysis. Muscular incoordination and, less frequently, sensory changes are manifested. Cranial nerve involvement occurs in ascending, anatomical order. Death is usually the result of respiratory failure. Fatalities usually occur within the first week of disease, but are rare if the tick is removed before bulbar symptoms appear. A list of clinical findings would include: dyskinesia, ataxia, dysmetria, dysphagia, and muscle paralysis. These signs are of varying severity and usually bilaterally symmetrical. Occasionally localized muscular involvement results.[7, 8] Spinal fluid changes are usually lacking and laboratory tests are of little positive aid in diagnosis. Body temperature characteristically remains normal except for occasional terminal elevation.

Tick paralysis is an acute, afebrile, rapidly progressive syndrome associated with tick attachment. Neurological symptoms are manifested without spinal fluid or muscle changes. These findings help differentiate poliomyelitis, Guillain-Barre syndrome, and muscular dystrophies which have many common symptoms.

The complete removal of all ticks, with careful extraction of the beak, constitutes adequate therapy in the majority of cases. Spontaneous recovery occurs if the tick completes engorgement and detaches before paralysis is advanced. When bulbar symptoms are present, proper care may necessitate the use of respirator, aspirator, parenteral feeding, and such other symptomatic procedures as are deemed necessary. There is no specific antidote for the suspected toxic substance.

CASE REPORTS

Case No. 1, O. D. A 7 year old Negro female had been asymptomatic and normally active until September 5, 1949. Thirty-six hours before hospital admission, an altered gait was noticed by her mother. At bedtime, three hours later, the child was unusually tired but had no other complaints. On the following morning, she was extremely weak and unable to arise from bed. She was conscious and apparently rational although listless. During the three days before this illness, the patient had been at home in Monroe, Louisiana. In the preceding eight days, she had resided on a small farm near Calhoun, Louisiana (Ouachita Parish).

Upon hospitalization in Monroe, examination revealed normal temperature, generalized muscular weakness, incoordination of upper extremities, loss of deep reflexes in both lower extremities, and bilateral horizontal nystagmus. A lumbar puncture was performed revealing clear fluid, normal dynamics, no cells, and a protein determination of 11 mg. per cent. Progression of symptoms occurred during the next eight hours of observation. The patient then was transferred to the Charity Hospital at New Orleans.

The child was well developed, well nourished, and presented no evidence of dehydration. Her pupils were small, but round, regular, and equal. Extraocular muscles functioned normally and the previously described nystagmus could not be demonstrated. The eye grounds appeared normal bilaterally.

The child was extremely listless and lethargic. She responded slowly but accurately to questioning. Generalized, symmetrical, flaccid paresis was evident without accompanying muscle spasm. Weakness was especially marked in the flexor, adductor, and abductor thigh muscles. The legs could be elevated only with great difficulty. No evidence of discrete abdominal musculature weakness was elicited. Head drop existed with upper extremity weakness, dysmetria, and marked lack of coordination. The intercostal and cranial nerves appeared unaffected and no respiratory or cardiac distress was witnessed. The patient was unable to lift her head or pull herself to a sitting position.

There was suppression of both deep and superficial reflexes. Ankle and knee jerks were barely obtainable and the abdominal reflexes were absent. Deep reflexes in the upper extremities were hypoactive and coarse intention tremor with past pointing and adiadokokinesia were evident. All findings were bilaterally symmetrical.

No gross sensory changes were present and subjective symptoms were lacking entirely. There was no evidence of meningeal irritation. No pathological reflexes were demonstrated. The child's serious condition caused no apparent apprehension on her part. At no time during this illness was fever recorded.

Lumbar puncture showed clear fluid under a pressure of 140 mm. of water. Manometric studies were normal. The Pandy reaction was negative and there were 2 lymphocytes per cu. mm. Culture was negative. Quantitative protein was 31 mg. per cent, glucose was 55 mg. per cent, and chlorides were 774 mg. per cent. Flocculation and

complement fixation tests for syphilis were negative. There was no pellicle formation.

The Mantoux test (1:1000) was negative. Urinalysis findings were within normal limits. The leukocyte count was 7650 with 79 per cent neutrophils, 5 per cent eosinophils, and 21 per cent lymphocytes. The erythrocyte count was 5 million with 14 Gm. of hemoglobin. Blood Kolmer and cardiolipin tests were negative.

Upon initial examination at Charity Hospital, a partially engorged tick (approximately 1½ by 2 cm. was discovered in the right parieto-occipital region. This was removed *in toto* and later taxonomically identified as a nongravid female *Amblyomma maculatum*, the Gulf Coast tick.*

Four hours after removal of the tick, the patient was able to hold her head erect and, with aid of hands, rise to the sitting position. Past pointing and an intention tremor were less marked. After ten hours, the child was considerably more alert to her surroundings and interested in other children in the ward. Her deep reflexes were more active and return of muscle power was noted in both lower extremities. Within twenty-four hours after removal of the tick, the child sat spontaneously, was able to stand, and walked with moderate ataxia. Neurological reflexes were physiological.

Within thirty-six hours ataxia was less noticeable, and within forty-eight hours all abnormal findings had disappeared and the patient was ready for discharge.

Case No. 2, M. M. An 8 year old Negro female, resident of Franklinton, Louisiana, became ill on the evening of September 13, 1949. She complained of vague dull aches and numbness in all extremities. The following morning these symptoms were more marked and were accompanied by generalized headache. After a light breakfast, she vomited once. By noon of that day, the child had difficulty in walking, coordinated poorly, and by late afternoon was unable to pull herself upright. Persistence and progression of symptoms continued through the following day (September 15). By this time, the child was extremely weak and unable to care for herself, yet remained well oriented.

Fever was thought not to be present but temperature recordings were not made.

On September 16 and 17 the child was very tired and listless. The parents described no change in her general condition. Loss of muscle power and other signs apparently failed to progress and bulbar symptoms were denied. The child remained at home during this phase of illness.

On September 19, the parents discovered a large, engorged tick embedded in the scalp. The approximate site of attachment was over the region of the posterior fontanel. The tick was removed intact and destroyed without being identified.

After removal of the tick, the child slept for slightly over two hours, and upon awakening found that she could pull herself upright and could stand. Gross weakness continued but twelve hours later the patient was able to walk.

Approximately twenty hours after the tick was removed the child was brought to Charity Hospital where examination revealed moderate ataxia and dyssynergia. The patellar, achilles, and abdominal reflexes were bilaterally absent. Hyporeflexia and incoordination were evident in both upper extremities. Actual muscle paresis was minimal and not well localized. Fever was absent throughout hospitalization.

The cerebrospinal fluid was colorless with an initial pressure of 140 mm. of water and normal dynamics. The fluid contained 3 lymphocytes per cu. mm., 90 mg. per cent of total protein, 54 mg. per cent of glucose, and 800 mg. per cent of chlorides. The Pandy test was negative. The Kolmer and cardiolipin tests were negative and no decolorization was seen in Lange's colloidal gold test. No bacterial growth occurred and a pellicle failed to form.

The correctness of the original protein determination was questioned and a spinal puncture was repeated the following day. Again the fluid was clear and dynamics were normal. There were 4 lymphocytes per cu. mm. Chemical analysis showed the presence of 9 mg. per cent of total protein, 50 mg. per cent of glucose, and 740 mg. per cent of chlorides. Pandy's test was negative. No organisms were cultured from the spinal fluid.

Blood cardiolipin and Kolmer tests were negative as was the Mantoux test (1:1000). The leukocyte count was 8800 with 39 per cent neutrophils and 61 per cent lymphocytes. There were no eosinophils. The erythrocyte count was 4.6 million and the hemoglobin 13.5 Gm. Blood was sterile to culture and the urine was within normal limits.

Forty hours after removal of the tick the child had no complaints, walked without an altered gait, and reflexes had returned to normal. She was discharged, apparently well, after three days in the hospital.

DISCUSSION

Although this is only the second report of tick paralysis in Louisiana, the disease may be considerably more prevalent than is realized. Its frequent termination in death when unrecognized and untreated constitutes adequate reason for alerting physicians to its existence. Any case of unexplained, acute flaccid paresis should demand consideration of tick paralysis. Further study and description of cases may help to elucidate the obscure pathogenesis.

Amblyomma maculatum, the tick associated with case No. 1, has not been cited previously in connection with tick paralysis al-

*Appreciation is extended to Mr. E. B. Johnson, Louisiana State Board of Health entomologist at Monroe, for identifying the tick.

though it is widely distributed along the Gulf Coast. In Louisiana, it occurs across the southern part of the state and is unusual in the northern parishes where this patient apparently became infested.

Two of the other three ticks associated with tick paralysis in this country also have been found in Louisiana. *Amblyomma americanum* is distributed widely in this state. This species has been associated with a case of tick paralysis from Rapides Parish.[9] *Dermacentor variabilis* is abundant in the northern part of the state and is found in small numbers in the central and southern parts. *Dermacentor andersoni* has not been reported from Louisiana, but is responsible for many cases of tick paralysis in other areas.

SUMMARY

Two cases of tick paralysis are recorded from widely separated locations in Louisiana. In one case the tick was identified as *Amblyomma maculatum*, which has not previously been implicated in this syndrome.

These cases are reported to alert physicians to the occurrence of tick paralysis in Louisiana. The disease is characterized by generalized flaccid paresis and may result in death unless the offending tick is detected. Prompt and complete recovery usually follows removal of the tick.

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WEAKNESS OF THE LOWER EXTREMITIES AS A COMPLICATION OF PREGNANCY*

HILMER W. NEUMANN, M. D.†

NEW ORLEANS

Weakness of the lower extremities, as a complication of pregnancy, has been recognized for many years. It is a disturbing clinical entity that has received scant recognition in obstetrical textbooks. This complication falls within the province of the obstetrician but is often baffling because of the neurological implications. On the other hand, the neurologist sees but a small number of these cases and has, therefore, tended to regard the condition as a rare complication of pregnancy and labor.

Generalized weakness of one or both lower extremities following labor is more common than is generally realized. This paper records 3 cases at the Henry Ford Hospital and reviews recent additions to the literature relating to the etiology of this condition.

In a previous communication[1] the differing etiologic factors leading to this malady were discussed. These basically are: emotional (hysteria); vascular disorders in the brain; bone change (osteomalacia, caries); muscle disorders; infections, local or general; and the trauma of childbirth.[2]

CASE REPORTS

Case No. 1. Mrs. H. B., a gravida 3, para 2, was admitted to the obstetrical service of the Henry Ford Hospital in March 1947, and was delivered by low forceps extraction of a 3990 gram infant from an L.O.A. position after an uneventful nine hour and twelve minute labor.

The pelvic measurements were: interspinous 25 cm., intercristal 27 cm., external conjugate 18 cm., biischial 11 cm., and the anteroposterior diameter of the outlet was 9.5 cm.

The fetal head measured 10 cm. in the occipitomental diameter, 12 cm. in the occipitofrontal, 11 cm. in suboccipitobregmatic, 10.5 cm. in the biparietal, and 9 cm. in the bitemporal.

There was a history, obtained from the patient,

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†Formerly of the Henry Ford Hospital, Detroit, Michigan. Present address, 2700 Napoleon Avenue, New Orleans, Louisiana.

of having struck the lateral aspect of her right knee on the day prior to delivery. Soon after delivery, a foot drop was present on the right. There were no objective sensory abnormalities of the right leg or foot. The ankle was reduced on the right.

One week later there was minimal peroneal action present but no action of the tibialis anticus or extensor muscle on the right.

The patient was again admitted in September 1947, at which time no mention of a foot drop was included in the history.

Case No. 2. Mrs. I. La G., a gravida 3, para 2, was admitted to the obstetrical service of Henry Ford Hospital in June 1947, and was delivered by midforceps extraction of a 3550 gram infant, which had been rotated from a R.O.T. position to an R.O.A. position, after a twenty-five hour and forty-nine minute labor.

X-ray pelvimetry measurements were: True conjugate 11 cm.; transverse diameter of the inlet, 12.5 cm.; anteroposterior diameter at midpelvic 11.1 cm.; interspinous diameter of 11.1 cm.

The average fetal head diameter was 10.5 cm. and the greatest diameter was 13.5 cm.

The foot drop in this case occurred on the seventh postpartum day and was on the left side. There was loss of action of the tibialis anticus and extensory loss on the plantar aspect of the left foot.

The patient was seen one month later, at which time there was no residual paralysis or sensory loss.

Case No. 3. Mrs. C. W., a gravida 1, para O, was admitted to the obstetrical service of the Henry Ford Hospital in March, 1948, and was delivered by midforceps of a 3800 gram stillborn infant from a persistent R.O.P. position after a forty-nine hour and ten minute labor.

The x-ray pelvimetry measurements were: true conjugate, 11.4 cm.; transverse diameter of the inlet, 11.9 cm.; transverse diameter of the midpelvis, 10.4 cm.; anteroposterior diameter of midpelvis 11.5 cm.; posterior saggital 8.8 cm.; intertuberous 10.6 cm. X-ray interpretation mentioned a "very prominent promontory."

The fetal head measured 15 cm. in the occipitomental diameter, 13 cm. in the occipitofrontal, 9 cm. in the suboccipitobregmatic, 9 cm. on the biparietal, and 8 cm. in the bitemporal.

On the first postpartum day, the patient was unable to abduct, adduct, or extend the left foot. This was accompanied by a "burning sensation" on the left lateral aspect of the leg and foot as well as the dorsum of the foot. There was "numbness" and "tingling" in the same areas.

Neurological examination showed gluteal weakness, as well as weakness of the adductors and abductors of the left foot. There was hamstring weakness and calf muscle weakness, accompanied by complete loss of the tibialis anticus and posterior and peronei on the left. Hypesthesia to pain and temperature was present on the dorsum of the foot and lateral aspect of the leg. A typical steppage gait was present.

She was again seen in November 1948, at which time the steppage gait was still present, as was the inability to dorsiflex or adduct the foot. Sensory impairment was not present.

SUMMARY OF LITERATURE

In 1854, Churchill[3] gathered from the literature and his own practice 34 cases of paralysis occurring during pregnancy and labor. In 1867, Bianchi[4] described the "traumatic paralysis of the lower extremities of women recently confined." In 1867, LeFebvre,[5] devoted his thesis to a minute anatomical study of the sacral plexus and later, Fere (1879),[6] Arnould (1892)[7] undertook the study again. The anatomical basis of the paralysis was elaborated by Hunermann[8] in 1892. Vinay (1894),[9] Leclerc (1905),[10] and Patel (1905)[11] published new cases which they presented considering the question again and discussing the anatomic and pathogenetic data.

The work of Hunermann placed emphasis on the fact that the weakness almost always affected the muscles supplied by the common peroneal nerve. Hunermann felt that injury to the peroneal nerve occurred at its point of origin in the lumbosacral cord. The dorsal portion of that cord is compressed between the lateral wall of the pelvis and any mass descending through the pelvis. Compression of the fibers is considered the etiologic agent and the fetal head or forceps is the compressing agent.

Thomas[12] reported three cases, these occurring (1) in a normal pregnancy, (2) in difficult labor and instrumentation, and (3) in a large child in a contracted pelvis. In one case, symptoms of nerve root compression appeared before instrumentation. In one case, no such symptoms appeared. A case of bilateral injury was explained on the basis of reapplication of the forceps with injury to both trunks.

Bardeen,[13] of the Anatomy Department of Johns Hopkins University Medical School, made 200 dissections of the sacral plexus and found it not unusual that the peroneal and tibial nerves were separate up to their origin from the sacral plexus. He suggested that injury to the lumbosacral

cord, causing motor and sensory dysfunctions so sharply limited to distribution of the peroneal nerve, as occur in the obstetrical paralysis, could be explained on the basis that branches of the roots that form the peroneal nerve are given off from the dorsal aspect of the plexus and lie next to the bone, while those forming the tibial nerve lie on the muscle and, therefore, the dorsal offshoots would be more likely injured.

Kleinberg[14] subscribed to the pressure theory that qualified in it an attempt to explain unusual cases. He maintained that one or more factors are always present in this complication of childbirth: disproportion, prolonged or difficult labor, or instrumentation. As a result of any one of them, an abnormal increase in intrapelvic pressure causes the lesion, unilateral or bilateral. In a like manner, a case described by Patel, in which a typical paralysis developed in the six months of pregnancy, was explained on the basis of hydramnios producing the increased intrapelvic pressure.

There are on record cases of peroneal neuritis following normal deliveries. Churchill stated: "If we recollect the number of severe and prolonged instrumental deliveries which take place without any such results (as well as uncomplicated cases which develop the condition), .I think we must respect this peculiarity of labor as a necessary or frequent cause." He offered no alternate explanation.

An interesting relation between the position of the presenting vertex and the side of the paralysis was noted by Mills[15] in a review of 6 cases. In 5 of 7 cases, the paralysis appeared on the opposite side of the greatest diameter of the fetal head, and she attributed the paralysis to trauma during instrumentation. She was upheld in her opinion by Beattie,[16] who studied 33 cases.

Injury by instrumentation, however, does not seem to be the only explanation for the paralysis. Compression of a nerve produces interruption of its conductivity, which is usually temporary, the severity varying directly with the force and duration of the pressure. During forceps delivery, pressure on the lumbosacral cord by forceps per se would be relatively short. It is improbable that the forceps directly caused the trauma to the nerve trunk and the paralysis, unless much force is used, which then should result in injury to the birth canal and other pelvic tissues.

Lambrinudi,[17] referred to the paper by Brooks, presented before the Guy's Hospital Anatomical Society, in which the latter reported on the dissection of twenty pelves of women who died shortly after a labor which was not difficult. These observations revealed that during the first stage of labor there is a rotation backwards of the sacrum (the axis of this rotation being through the lower half of the articular facet). In some instances this rotation is as much as one-quarter inch. Since the rotation occurs to enlarge the anterior-posterior diameter of the pelvis, it is not difficult to perceive that in difficult labor the displacement would be even greater. This, coupled with the fact that the lumbosacral cord is known to be one of the tautest nerves in the body led to the suggestion that the mechanism of the palsy is traction on the lumbosacral cord as the sacrum rotates backward. This would locate the lesion in the roots of the nerve rather than along its course. This theory would account for the antepartum sciatic pain which occurs frequently in gestation, as well as the rare postpartum paralysis. Although he saw no cases of his own, Lambrinudi believed that the postpartum paralysis and the antepartum sciatica were explainable on this common basis.

The theory of Lambrinudi is logical; traction on the cord producing root injury would explain the cases of bilateral paralysis. Such an explanation, however, does not account for the greater frequency of unilateral cases.

In an attempt to explain unilateral cases, Duncan,[18] in 1921, elaborated the theory of the injury being due to pressure on the common peroneal nerve from an external source. If one flexes the knee to a right angle, and makes pressure just behind the insertion of the tendon of the biceps into the head of the fibula and on the posterior

surface of the fibular head, the nerve is easily located, especially if there is little subcutaneous fat. It is at this point that the nerve is especially vulnerable to injury. The injury may be by pressure of the stirrups, in which the patient is placed for delivery. It may likewise be due to injury caused by rotation of the head of the fibula in its proximity to the nerve.

O'Connel,[19] has attempted to explain the paralysis on the basis of intervertebral disc disorder. One hundred and twenty cases of protruding lumbar intervertebral disc proven by operation have been tabulated by O'Connel. Twenty-three per cent of the cases were female. Of the female, 23 per cent had borne children, and of that number 27 per cent had recurrence of symptoms following labor or during pregnancy. The assumption is made that a correlation exists between the physiological changes of the organism during gestation and the occurrence of the symptoms of protrusion of an intervertebral disc. The high percentage of recurrence of symptoms following gestation supports the correlation. The Hunermann theory complements O'Connel's hypothesis; the back rotation of the sacrum through the relaxation of the normally rigid sacroiliac articulations. O'Connel suggests that analogous relaxation is highly probable in the intervertebral articulations.

Investigations by Abramson, Hurwitt, and Lesnick[20] show that the relaxation of pelvic joints is normal during pregnancy. Such relaxation has occurred early in pregnancy, which militates against a mechanical etiology. Relaxation of the symphysis pubis and sacroiliac joints occurs less frequently in primiparae than multiparae. Occupations necessitating exercise predispose to greater separation as do constitutional disease, rickets, and cachectic states.

Although there are no direct measurements to prove the laxity in intervertebral articulation during pregnancy, it may be assumed that such should occur. The symptoms recorded in these patients following labor parallel in some respects those of disc herniations.

Relaxation of joints occurs in the middle portion of the second trimester and is thence progressive; widening of articulations has been demonstrated as early as the eighth week.

When protrusion of a lumbar disc occurs during pregnancy, three types of clinical syndrome may follow. There may be the development of backache and sciatic pains as described by O'Connell. At times, paralysis has developed during or after labor and there has been no associated pain. In the majority of reports on maternal obstetrical paralysis, pain has been stated to have been present but, in some, no mention is made of it. Finally, there is the majority group in which the disc protrusion gives rise to both sciatic pains and muscle weakness.

The work of O'Connell has revived interest in this possibility in the puerperium and the number of recorded cases of protruded disc is increasing. Investigation should be undertaken to determine the condition of the intervertebral articulations during the latter months of gestation.

Recent work from France opposes the disc theory of O'Connell. Trillat and Dumont[21] agree that a vertebral lesion is certainly possible during a spontaneous or instrumental delivery. Nevertheless, these investigators believe that the "aggravation of symptoms in relation to discal hernias observed during pregnancy is easily explained by the relaxation and absorption of the disc during pregnancy (but) this mechanism scarcely seems preferable to one which provokes the sudden appearance of a unilateral paralysis in a pregnant woman and appears certainly more traumatic and more violent."

Trillat and Dumont have also observed 5 cases of the paralysis during those years corresponding with the severe dietetic restrictions of the war and noted that vitamin B therapy administered in 4 cases gave excellent results with cure in two or three weeks. They question the possibility of hypovitaminosis accompanying compression as the cause of the syndrome. In one of the cases the patient had delivered a 3600 gram infant resulting in a peroneal palsy two

years prior to her current pregnancy. She began oral vitamin therapy during the sixth month of the second pregnancy, and, although she suffered cramps and pain in her left thigh, leg and toes during the delivery of a 3450 gram infant, she developed no paralysis.

It seems more logical to the author that the poor diet of the war years in Europe predisposed to general cachetic states, which, if present in a woman, would naturally affect the musculature of the true pelvis. In such cases the lumbosacral cord would certainly be more vulnerable, if that should happen to be the etiologic thesis to which one subscribes.

In spite of the volume of cases presented by investigators prior to the work of O'Connel, the conclusions of the latter are indisputable. His statistics are upheld by operation, and the correlations presented are entirely scientific. He is upheld by Lantuejoul, who believes that the mechanism of disc herniation at L4 and L5 "seems more probable than a compression of the nerve or roots by the fetal head."

With the advent of the hypothesis of O'Connel, it would seem logical that the proponents of this work would make sincere efforts to prove disc herniation in the suspected cases. We regret that such has not been done in the 3 cases reported in this paper or in previous investigations[1] by this author.

We have attempted to put forth the various scientific factors interrelated in these cases in an effort to show the possible correlation between intervertebral disc relaxation (supposed) and the known phenomena of pelvic and sacroiliac relaxation. Although we have not been as detailed as in former summaries,[1] we have, nevertheless, tried to acquaint our readers with the present status of this interesting, if not copiously studied, condition.

SUMMARY

A review of the historical literature and recent advances regarding maternal obstetrical paralysis has been made.

Theories concerning the etiology of this condition have been reviewed.

There may be an increased risk of lumbar intervertebral protrusion during gestation. This may be due to changes in the intervertebral points analogous to those known to occur in the pelvic joints during pregnancy. Recent investigation from France proposes that in addition to any one of the various etiologic factors thus far theorized, the presence of severe malutrition and hypovitaminosis may predispose to this condition.

A plea is made for further scientific investigation in this field, in order to more significantly prove or disprove the lumbar disc theory.

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MANAGEMENT OF RECENT NASAL INJURIES*

FRANK L. BRYANT, M. D.
SHREVEPORT

Trauma to the nose may involve only the skin and subcutaneous tissues or it may be severe enough to either fracture or dislocate part or all of the bony and cartilaginous framework. In the period from January 1, 1947, to October 1, 1949, 76 patients

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who had suffered some form of nasal injury were seen in the Section of Rhinolaryngology and Plastic Surgery of the Willis-Knighton Clinic. Of these, 37 had a fracture of the nasal bones and the remaining 39 had some injury to the skin and subcutaneous tissues. Those with simple abrasions were not included. The patients whose records comprise this report were seen from within a short time after the injury to a two week period. Those who came in after fourteen days were considered as neglected nasal injuries. This is a special problem which deserves its own analysis and conclusions.

A patient with an injured nose is initially concerned with the cosmetic result. The rhinologist, notwithstanding his desire to restore or even improve the original contour, is primarilly concerned with and interested in restoring nasal function. He is aware that the relationship between anatomy and physiology is a most intimate one. In other words, normal architecture is essential for normal function. An uncorrected nasal injury not infrequently leads to nasal occlusion of one or both nasal cavities; also, changes in the pressures and directions of the air currents in the nasal cavities will often result. Abnormal contacts between the septum and turbinate will frequently produce syndromes of headache, and neuralgia, by direct pressure or by interference with the drainage of the nasal secretions. The relation of the nasal (and sinus) cavities to speech should likewise not be overlooked. Sound vibrations originating in the larynx have the benefit of this resonating chamber. Resonant sounds are improved to full quality by free vibration of the air within these cavities.

The importance of a careful examination cannot be overemphasized. Many patients are surprised to learn that a seemingly slight blow can produce a fracture or dislocation, especially in a younger individual. The primary reaction about the nose in a patient within the first decade of life is usually greater than in an adult. This may be often misleading by disguising the fracture unless a systematic and careful examination is made.

During this period, 76 patients were seen with some form of injury to the soft parts or the bony framework, or both. The immediate treatment of the injured nose is the control of bleeding with appropriate measures. An emergency suture may be necessary to control active arterial flow; gelfoam or oxycel used in conjunction with gauze dressings may be sufficient to suppress oozing until adequate repair can be done. Injury to the skin and subcutaneous tissues with no evidence of fracture should still receive careful attention. Faulty cleansing of tissues, irregular approximation of skin edges and coarse suture material will result in an unsightly and often disfigured nose. The wound should be cleansed with peroxide and liquid soap or a detergent so that all foreign substance is removed. A small even edged tooth brush is advantageous to loosen dirt particles embedded in the tissues. Lacerations about the nose and cheek are frequently irregular. Anchor sutures placed at critical intervals before actual suturing is done will permit a more exact and even approximation. A subcuticular type of suture can frequently be employed in certain kinds of lacerations about the nose and face if the cut is at right angles to the skin surface. Only the finest plastic suture needles and material should be used. Early removal of the sutures followed by a gauze mesh collodion dressing will produce a less obvious scar. If trauma was so mutilating that skin was lost at the time of injury or must be sacrificed because of shredding, there is no contraindication in doing an immediate repair by using a full thickness skin graft from the post auricular area. The sutures, preferably of silk, should be long enough to tie with their opposites over a pledget of sterile mechanics waste with a piece of vaseline gauze interposed between it and the skin graft. A successful take can be expected if the defect is not too large and the bed is clean. In many patients in whom no injury to the bone can be determined, a definite subperiosteal hematoma can be felt. In the larger ones

aspiration of the blood, if recent, by needle can be done.

Strips of adhesive should be applied firmly over which is tightly placed a metal splint. This will hasten recovery and give protection and comfort to the patient from additional minor irritation. Such a dressing will minimize or obviate the formation of a hump due to the organization of the hematoma.

A fracture of the nasal bones may exist alone or be associated with fractures of the facial bones. Obviously, fractures elsewhere may also exist. Intracranial disturbance may also be present. The sequence of the management must be determined by the attending surgeon. Naturally, shock and concussion, if present, should receive first consideration. First aid and supportive measures to the nose which will not detract from the general condition, should be immediately carried out. Deeply lacerated tissues should be protected until a more satisfactory examination can be made. Any isolated pieces of cartilage or bone should be cleansed and replaced until they can be realigned more correctly, later. Satisfactory results can often be attained by utilization of seemingly fragmentary pieces of the osseous framework. Careful intranasal packing to replace and reposition any separated mucous membranes into normal position is important so that adhesions and stenosis will not subsequently develop.

SIGNS AND SYMPTOMS

Hemorrhage: It may come from one or both nasal cavities. It is present in the more severe kinds of fractures of the nose. It may become so profuse that intranasal packing is necessary, and immediate repair of the fracture, therefore, is delayed. Gelfoam or oxycel applied to the bleeding area and held in place by gauze packing is usually sufficient to control this type of hemorrhage.

Ecchymosis and Swelling: They are due to extravasation of blood into the subcutaneous tissues. Depending upon the site and severity and type of the trauma, the extent of the ecchymosis will be determined. The height is usually reached in from forty-eight hours to seventy-two hours. It gradually subsides in a week to ten days.

Nasal obstruction: It may be due to the swelling of the extravasated blood against the upper lateral cartilages, or to hematoma beneath the mucous membranes. It may be due to the deformity of the nose itself, either by the fracture of the nasal bones or the displacement of the septal cartilage or both.

Pain and tenderness: They may be due to the external lacerations or by the impingement of a broken fragment against the nerve. Palpation or manipulation in the examination will usually be painful if not existent before.

Deformity: It will be present if swelling, displacement, distortion or depression exists. If the edema and ecchymosis are marked, as is not unlikely in children, it may disguise any bone injury.

DIAGNOSIS

It is well to consider any nasal trauma as potentially a fractured nose until a careful examination has indicated otherwise. Tenderness especially over one portion of the nasal pyramid, swelling, ecchymosis, hemorrhage, all suggest nasal fracture. If no external deformity exists a positive diagnosis may only be inferred. Roentgen rays may then give a positive diagnosis. Gross deformity with crepits makes the diagnosis obvious and the roentgen ray merely corroborates the clinical findings.

The following paragraph is taken in part from Cottle and Loring. It is an excellent description of the anatomy.

"The external nasal pyramid from a surgical point of view is divided into the bony vault, the cartilaginous vault, and the lobule. The latter is further subdivided into the tip, alae, and the columella. The bony vault is made up of the nasal bones and the frontal processes of the superior maxilla united in the midline onto the perpendicular plate of the ethmoid. In young individuals the plate extends anteriorly beyond the smaller nasal bones. Attached to the under sides of the bony vault are the upper lateral cartilages. These in reality are the wings of the septal cartilage of which are an integral part and together which constitute the cartilaginous vault. In each side of the lobule is the horseshoe shaped alar cartilage commonly called lower lateral cartilage. The columella extends in the midline from the tip of the nose to the lip. The free end of the septum

pushes into the dorsal half and extends to the superior nasal spine of the superior maxilla."

ANESTHESIA

To properly repair the injured nose either for lacerations of the soft parts or to reset the bony pyramid, good anesthesia is necessary. Whether general or local anesthesia is to be used is obviously dependent upon the judgment of the surgeon as he considers the age and condition of the patient and the extent of the injuries, and the time of the repair in relation to the time of injury. In those patients in whom debris and foreign substance are present, general anesthesia is preferred to permit thorough cleansing.

CLASSIFICATION

1. Incomplete
2. Linear
3. Unilateral depression
4. Bilateral deviation
5. Bilateral depression
6. Fracture from below
7. Compound fracture with comminution.

INCOMPLETE

Four patients, all children, suffered this type of fracture. Ossification is incomplete in this first decade of life. Swelling is usually localized and may also be visible intranasally. A roentgen ray taken from above downward so that contrast can be visible for a partial break is important. To correct this type of fracture a general anesthetic, preferably vinythene, is preferred. To attempt to manipulate the nose of a child with local anesthesia is a not too satisfactory procedure. The parts can be better aligned when the patient is asleep. Vaseline gauze packing is placed into the nasal cavity and a metal splint of soft copper covered with adhesive is fashioned to fit firmly over the dorsum of the nose. The packing may be removed in from forty-eight to seventy-two hours, but the external splint is kept on for at least five days.

LINEAR

This type of fracture presents the least serious problem. It was seen in 6 patients. There is but little swelling and nosebleed is common. Ecchymosis will be minimal. Tenderness directly over the site of the fracture may be usually elicited. A small firm external dressing should be applied to minimize the formation of a hematoma. Intranasal packing may be required although not always. If a hematoma has already formed it should be drained, if of any size, and packing inserted. The external dressing may require reapplication in two to three days and should be allowed to remain on for at least five days.

UNILATERAL DEPRESSION

This type of fracture occurred in 10 patients. It results from a forceful perpendicular blow to one side of the dorsum of the nose. The nasal bone is driven downward and inward and therefore separated from its fellow and the frontal process of the superior maxilla. Deformity is obvious. Before attempting to correct the fracture it is important to determine by palpation and measurement the distance to insert the elevator to restore the depressed fragment to its normal position. If it is inserted too far the pressure will be exerted against the nasal process of the frontal bone and therefore be futile. The instrument should be inserted to a point just underneath the middle of the nasal bone so that the upward and outward pressure will be most effective. Careful intranasal packing is inserted into the nasal cavity on which the fracture occurred. An external splint of adhesive-covered soft metal firmly fashioned over the dorsum is applied. The packing may be removed in forty-eight hours and the external splint re-fashioned, but kept on for one week.

BILATERAL DEVIATION

(Elevation and depression). There were 8 patients who suffered this type of injury; 6 were under 16 years of age and the other 2 were adults. A severe blow against one side of the nose at less than the perpendicular direction will produce this type of fracture. The nasal bone receiving this impact is driven inward being separated from its fellow and the nasal process of the frontal bone and the frontal process of the superior maxilla. The other nasal bone is driven outward. The septum, especially the mesoethmoid portion, may easily share in the trauma. Tearing of the mucous membrane is also not unlikely. Roentgen rays

readily show this type of deformity. Before any attempt to replace or realign the broken nose is made, good anesthesia is imperative. The depressed bone is replaced into position in much the same manner that was used in the unilateral depression. By firm thumb pressure the opposite one can be depressed into its lower and normal place. Vaseline packing is carefully inserted into each nasal cavity. An external dressing of dental stent compound is applied so as to exert pressure especially on the sides of the nasal bones to maintain a narrow bridge. This external dressing should be reapplied in a few days when some of the swelling has subsided and should be allowed to remain for at least seven days. (Figures 1 and 2).

BILATERAL DEPRESSION

This occurred in 3 patients. When a severe force strikes the nose directly over the bridge, this type of injury results. The nasal bones are torn from their common attachments and driven downward and inward. The septum is also usually torn and broken; considerable tearing of the mucosa is found. The saddle type of nose will result if correction is not made. Internally sequestration, abscess of the septum, and adhesions may result. The surgeon should be able to palpate the cleft over the bridge, the width of which is dependent upon the amount of separation and depression of the nasal bones. Because local anesthesia has not been found to be satisfactory for the repair of this type of injury, it is better to use general anesthesia. A postnasal pack should be used as bleeding is frequently quite brisk when manipulation is done for correction. The septum may be difficult to be straightened especially if there is splintering; some pieces may have to be removed so that alignment can be done. Pink dental wax placed against the mucous membranes on either side of the septum will aid materially in maintaining its integrity. Vaseline gauze packing is carefully packed into each nasal cavity. This internal dressing should not be removed until the third or fourth day. The external dressing should be carefully reapplied so that

Fig. 2. Postoperative result.

Fig. 1. Bilateral deviation. Left nasal bone is depressed and right nasal bone is elevated.

the bridge is kept narrow. The external splint in this type of fracture should be kept on in some instances as long as two weeks if the injury was severe. (Figures 3 and 4).

FRACTURES FROM BELOW

There were 3 patients with this variety of nasal fracture. Two variations can occur or both may be present, from the force which comes from below. In one, the cartilaginous septum is displaced or torn loose from the maxillary crest or spine; it may also be fractured from this bone. In the second, the nasal bones are driven either above or below the nasal processes of the frontal bones. If they are found to be above the globellar angle is lost; and, if below, it is apparently widened. A clear concept of this type of fracture is imperative. Roentgen rays will materially aid in the diagnosis. Before any attempt is made to realign the nasal bones, they must be pulled downward into their normal field, after which they can then be repositioned into their normal place. Through the usually severely lacerated mucous membranes, especially in the superior aspect, an opening is afforded through which one may insert the grasping forceps to reposition the displaced nasal bones. As before, intranasal vaseline gauze packing carefully placed is important; also dental stent compound is preferred for the external dressing for this type of fracture. It is firmer and may be fashioned more accurately over the dorsum. It should be allowed to remain in place for at least ten to fourteen days in this type of fracture.

Fig. 3. Bilateral depression. Both nasal bones are depressed with flattening and widening of nasal bridge.

Fig. 4. Postoperative result.

COMPOUND FRACTURES

There were 3 patients who suffered compound nasal fractures. One was a girl of 16 years who was in an automobile accident; the second a boy of 8 years who was kicked by a mule (he also received a deep laceration of the cheek and compound fracture of the superior maxilla). The third was a boy of 10 years who was thrown from a horse. In each instance the normal nasal contour was completely destroyed; dirt and

debris were present and the bony and cartilaginous pyramid was in a comminuted amorphous mass. First aid and supportive treatment were given to each of the patients before any reparative surgery to the nose could be undertaken. No set rule can be given for repair. A thorough knowledge of the anatomy of the bony and cartilaginous pyramid is imperative. No fragments of bone or cartilage should be sacrificed, but they should be replaced and repositioned after meticulous cleansing. Careful suturing of the soft parts with plastic needles to minimize scarring is necessary. The external dressing should be so fashioned that after intranasal packing has been made, too much pressure will not be exerted to produce necrosis. The dressing should be changed frequently to watch for any stitch abscess and to keep it firm as the swelling and the endema subside. (Figures 5, 6, 7, 8).

Fig. 6. Profile view of same patient.

Fig. 5. Laceration of dorsum of nose which extends into both nasal cavities; left nasal bone partially comminuted; left upper lateral cartilage separated from nasal bone; septal cartilage lacerated.

Fig. 7. Postoperative result, face view.

Fig. 8. Postoperative result, profile view.

SUMMARY

Injuries to the nose may involve the soft parts or the bony cartilaginous framework, or both.

Injuries to the nose may produce fractures or dislocations which may be overlooked due to the hemorrhage and swelling.

Injuries to the nose should receive careful attention to restore both nasal function and nasal contour.

A series of 76 patients is analyzed and discussed. Approximately one half had some variety of nasal fracture.

THE MANAGEMENT OF COCCOGENIC DERMATOSES

JAMES K. HOWLES, M. D.*

NEW ORLEANS

There is no more troublesome problem in the field of dermatology than the care of the coccogenic invaders of the skin. To use a trite expression they are always with us. The reason for this great incidence of pyogenic conditions is easily explained when one considers the structure of the skin and the nature of bacterial growth. The skin is riddled with pores and follicles through which waste products, such as sweat and oily secretions, the products of the sebaceous glands, are exuded onto the skin surface from the underlying appendages of the skin proper; but in these indentations or invaginations are found myriads of bacteria of all kinds bacilli and cocci, as well as every other type of known organism, which find suitable growth requirements on the surface of the skin and in the various pores and orifices.

They are considered as normal saprophytes and do not cause any pathologic changes until they gain access, to the deeper or underlying tissue. The manner in which they reach these deeper tissues is varied, and yet easily understood. Trauma, such as rubbing and scratching, is the common cause. In fact, anything that produces an interruption in the normal contour of the skin will act as a point of entree for these normal saprophytes. Sometimes injury results in a break in the integument, but regardless of the nature of the trauma, or its severity, the result is an influx of these saprophytes. I frequently compare the change that occurs, due to the invading bacteria, to the activities of a group of boys in an unsupervised or undisciplined environment. The growing requirements are changes in these deeper tissues, and the previously innocuous saprophytic bacteria soon become pathogens, and may become a real and serious pyogenic infection with all the complications associated with such bacterial growth. This comparison and explanation is perhaps too simple, but it describes the changes which occur in the formation of many pathogenic organisms that gain entree from without through the skin.

Almost all micro-organisms are found normally on the human skin. Some remain as harmless saprophytes, but many others become pathogenic under suitable condi-

*From the Department of Dermatology and Syphilology, Louisiana State University Medical Center and Charity Hospital of Louisiana, New Orleans.

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tions. We are concerned only with coccogenic organisms in this paper; so we must limit our remarks to this group of micro-organisms. The cocci of concern to the dermatologist belong either to the staphylococcus or streptococcus groups.

Those pyogenic micro-organisms produce definite cutaneous diseases and often act as secondary or complicating factors in various other dermatoses. They usually, as stated, invade the skin from without and produce, as a rule, localized, pustular lesions, with or without tissue necrosis. Associated, constitutional phenomena may be present in many of these pyogenic infections.

TYPES OF COCCOGENIC DERMATOSES

The varieties of the coccogenic dermatoses are numerous and embrace too large a field for discussion here, but most of the cutaneous dermatoses included in this group belong to either the follicular or the nonfollicular group.

The follicular group comprises those diseases of coccogenic organisms that are localized in or about the hair follicles. They invade the perifollicular area or the hair follicle proper. Included in this group are the following follicular coccogenic dermatoses;

1. Furunculosis
2. Sycosis vulgaris
3. Carbunculosis
4. Impetigo of Bockhart
5. Folliculitis keloidalis

These follicular dermatoses are, as a rule, most difficult to treat because of the tendency of the cocci to invade the hair follicle, which frequently requires epilation of the hair shaft in the course of treatment. They are most prone to recur and usually require internal medications, as well as topical measures of therapy. Scarring, as a rule, is more common in this group of dermatoses, but is usually not of a deforming type.

The nonfollicular coccogenic dermatoses are that group which shows a tendency to involve the smooth skin with no particular predilection for the hair follicle or perifollicular areas. This group embraces quite a few entities including:

1. Impetigo contagiosa
2. Ecthyma
3. Abscess
4. Perlèche
5. Dermatitis repens
6. Dermatitis vegetans
7. Impetigo neonatorum
8. Chronic streptococcic ulcer
9. Dermatitis exfoliativa neonatorum
10. Pyogenic paronychia
11. Granuloma pyogenicum
12. Pyoderma gangrenosum

This is not a complete list, but includes most of the more common types of the nonfollicular varieties. Many of the dermatoses included in this group are autoinoculable, which means they are spread by self inoculation.

There is a miscellaneous group which may or may not show a predilection for the follicular or perifollicular structures. Chief among this group is erysipelas. Others to be included in this miscellaneous group are:

1. Intertrigo
2. Erysipelas
3. Gonococcus dermatitis
4. Gangrene of the skin
5. Cellulitis
6. Dermatitis gangrenosa infantum
7. Acute decubitus

Many of the coccogenic dermatoses are spread by scratching and rubbing; in other words, they are autoinoculable. These are of diagnostic importance, and since they are spread by one's self, will give a lead as to the cause and indicate the therapeutic procedure to follow. These autoinoculable dermatoses are usually secondary to trauma of some kind and have certain hallmarks useful in recognizing them. These are:

1. Appearance of lesions simultaneously on face, fingers, and genitalia. Their appearance is spontaneous.

2. Absence of lesions in inaccesible areas such as the intrascapular region.

3. Occurrence of lesions on both of two parts that may be brought into contact. These so-called intertrigenous dermatoses are found in such areas as the inner aspects of the arms and flank, the inguinal area especially, the contact surface of the penis and scrotum, as well as any points where

the surfaces of the skin touch or rub.

4. Appearance of lesions in scratch marks. Remember the human really claws as does a cat to relieve itching and the scratching is always in a lineal fashion. No one scratches in circles. There are a few doubtful criteria as in the case of lichen planus and psoriasis. Then too, one must not confuse the corymbiform stage of secondary syphilis with an autoinoculable factor. This secondary syphilide is characterized by one large papular or papulo-pustular lesion with numerous small satellite associated lesions. It resembles a nipple with enlarged periareolar lesions around it.

AUTOINOCULABLE COCCOGENIC DERMATOSES

1. Impetigo—the most superficial of the group.

2. Pyodermia—the intermediate stage.

3. Ecthyma—the deepest state. This last is really an ulcerated form of impetigo. These indicate the depth of the lesion and determine whether there will be any scarring associated with the eruption. It is a source of much comfort to a mother to be told that her child has impetigo or "Indian-fire" and that she need expect no scarring as a result of the eruption. Pyodermia is a simple pustular eruption and will scar in direct relation to the amount of the basal cell layer that is destroyed.

An illustration of this type of lesion is the pustule of acne vulgaris and of varicella, in which the amount of scarring depends upon the depth and extent of the particular lesion. In ecthyma there is always scarring, as this form of ulcerative impetigo is accompanied with marked destruction of the basal cell or germinating layer of the skin. Frequently, extensive loss of the overlying tissue follows the healing of the acute phase of the disease. Ecthyma is frequently caused by overtreatment of impetigo contagiosa or bullous impetigo.

It seems necessary here to discuss the impetigenous dermatoses, as this group of skin diseases is not clearly classified. The term impetigo requires a qualifying term such as contagiosa or bullous or follicular, to properly differentiate it. The old term "Indian-fire" is a colloquialism and is not used in all parts of the country, although the laity know it chiefly by that name. The high state of infectiousness of impetigo is not appreciated by most physicians. The hallmarks of the extremely common disease are autoinoculability, the superficial character of the eruption, the tendency to spread by very rapid peripheral extension with the formation of bullae or crusts or both.

The autoinoculable feature accounts for the distribution of the eruption on the fingers, the face, and at points of trauma, particularly the scratch areas. The acute crusted lesions have the appearance of being "stuck on" the skin surface. When the lesions become older they become indurated and there is usually a collarette of inflammation associated with each individual lesion. The rapid peripheral extension of the eruption is responsible for the circinate configuration and makes differentiation most difficult. There are usually successive exacerbations with the appearance of repeated crops of new lesions. When bullae are present the size of the individual lesions varies greatly and the contour of each lesion is not uniform. The crusted form of the eruption is the most common and the crusts are usually thin and very friable. These crusts are very easily detached from the base of the lesion. The problem of differential diagnosis is made less complex if these few important characteristics are considered.

Sycosis is the name applied to the pustular infections of the bearded areas. It is, of course, more common in men and the most difficulty encountered is in determining whether the pustular eruption is of coccogenic origin or associated with fungus infection. The treatment of the coccogenic type, or sycosis vulgaris, will be discussed later.

These infections of the hair follicles may be associated with complete destruction of the hair bulb or root of the hair. The initial lesion is usually a pustule or nodule with central softening. The feature that makes these hair follicle infections so recalcitrant to treatment is the depth to which the pyodermia extends into the invaginated area

of the follicle. In this country there are two types occurring in the scalp which are not too frequently encountered; these are the multiple peri-follicular abscess form and the folliculitis decalvans type; both varieties result in permanent baldness of a patchy type and scarring is a constant feature.

In the bearded area of the face a form known as follicular impetigo is very commonly seen and shows a tendency to become confluent. When the follicular type of impetigo occurs on the forarms and lower legs it is frequently called impetigo of Bockhart and is comparable to a sycosis of the bearded area of the face, but is usually very recalcitrant to treatment. When it occurs on the lower legs, the circulatory factor complicates the picture, especially in older individuals. Sycosis vulgaris differs chiefly from the follicular type of impetigo in that it involves the bearded region of the face and is usually discrete.

There is a form of folliculitis involving the face frequently resulting from close shaving and friction produced by rubbing and similar forms of trauma. This type of hair follicle involvement is sometimes referred to as folliculitis simple. The classification of various types of hair follicle involvement occurring in different areas of the body has a specific terminology. Most of the follicular and perfollicular pyodermias are, as previously stated, quite recalcitrant to the usual therapeutic efforts.

Many of the deep seated pyogenic infections extend into the sweat glands and give rise to chronic nodular inflammatory lesions which, as well may be expected, are very difficult to treat.

The purpose of this paper is to consider the method of approach in managing these coccogenic dermatoses; so we must limit our discussion to that end. It would be apropos to list the task at hand and to take up each procedure in order.

TREATMENT

The general principles of treatment of the coccogenic dermatoses are as follows:

1. Prevention of autoinoculation
2. Drainage
3. Reduction of bacterial population both superficial and deep
4. Correction of local resistance reducing factors
5. Correction of systemic factors.

The prevention of the autoinoculable factor is imperative if one is to succeed in clearing up these coccogenic dermatoses. Such a plan resolves itself into two procedures—the immediate course of preventing contact of infected surfaces by touching with fingers, and the avoidance of squeezing or traumatizing the infected areas.

Relief of itching is a must if one is to eliminate the incentive to scratch, with the resulting traumatic lesion. One must recall that most scratching is a reflex reaction and cannot be controlled entirely by will power. If one relieves the itching by the use of antipruritic and parasiticidal agents, much of the scratching will be eliminated. Antipruritics in the proper vehicle for these drugs such as cresote, weak phenol solutions, local anesthetic agents, such as nupercaine and procaine, are all useful at times. Many proprietary preparations, such as surfacaine and similiar preparations, either alone or in combination with other parasiticidal agents, are effective at times.

The treatment of parasitic factors either vegetable or animal is necessary if one is to be successful in treating these coccogenic dermatoses which are secondary in type.

The use of protective gloves and creams to reduce the irritation from scratching and to protect the covered areas from trauma caused by contact with infected areas should be used very often. I have found a good protective cream in "Pro-tek" which is used in many of the contact eruptions.

The immediate factors to be considered are the proper disposal and cleansing of such objects as contaminated dressings, wash rags, sponges, towels, and bath mats. The bedding and articles of clothing require special attenton. The frequent changing of diapers when required and the washing of such items as socks is not to be overlooked.

Contaminated slippers are a source of autoinoculation.

The number of cases one is called in to see in consultation for "Indian-fire" in nurseries, both in hospitals and other institutions, requires a definite plan of treatment. The technic to prevent contamination is as important as the actual therapeutic procedures employed.

In short, prevent autoinoculation by preventing contamination; relieve itching and overcome excessive dryness which often predisposes to pruritis and subsequent scratching. Protect the other exposed persons by adequate isolation, and above all discourage, and if necessary insist on, the prevention of visitors. One has only to visit the average private hospital nursery to see how imperative the no visiting rule is needed. The common practice of prolonged visits of grandmothers and grandfathers to say nothing of the influx of admiring aunts and uncles during nursing hours should be definitely discouraged. The reaction to some of these rigid rules is quite interesting to say the least.

Drainage: The fundamental principles of surgery apply to these coccogenic areas. The use of wet dressings to localize the exudate process precedes the necessary drainage. The rule of evacuating encapsulated or walled-off lesions as applied to ordinary surgical problems is to be used. The bullae and vesicles are to be trimmed when possible to enhance the drainage from the base of the lesions. It is advisable to clip them rather than rub them. Tearing and pulling of overhanging margins leads to further pustular infections. The use of sharp, small, curved scissors is recommended. The removal of hair and other follicular obstructions is advisable. The removal of comedone plugs, when possible, is conducive to better drainage.

A complete technic for the treatment of furuncles is not necessary here but a few points concerning these lesions may be necessary. It is inadvisable to lay hands on a furuncle until you have considered what it is. Determine if the usual staphylococcal infection is causing the follicular and sebaceous gland infection. If it is localized, do nothing that will extend it. Avoid breaking the leukocytic wall surrounding it, if possible. Remember the contents of the pustule are infectious as is the overlying skin which has a high bacterial content. Attempt to reduce it and, above all, avoid autoinoculation. The mere appearance of a furuncle indicates reduced general resistance and certain predisposing factors. Attempt to find these factors and prevent a siege by dealing with them. Always treat the furuncle conservatively and in most cases expectantly.

Consider the location of the lesion. Lesions of the nose, nasolabial fold, the lips and periorbital areas have an added danger because of the venous drainage to the meninges from some of these areas. Carbunculosis may result from lesions on the back of the neck, especially in older people and those with a diabetic history. Scarring and disfigurement, particularly of the face, neck, and shoulders, require some consideration. In the axillae, spread and dissemination is quite common, and sometimes unavoidable. In the fatty regions, such as the buttocks, infections of the subcutaneous tissue often ensue with the development of cellulitis and multiple sinus formation.

Too rapid incision often leads to prolonged infection. It is often wiser to let the lesion mature and point, or even drain itself. Lifting the top from a very early pustule may shorten the course of the infection. Avoid too radical procedures in treating cutaneous lesions. If extensive slashing and deep, cruciate incision is indicated it probably needs surgical consultation anyway.

Never squeeze, for to do so closes the drainage opening and favors the spread of the enclosed infection into the surrounding tissues. This procedure is to be definitely discouraged in the areas adjacent to the nose and lip. We advocate evacuation of ripe furuncles but discourage too radical procedures.

Painting the surface of early furuncles with cauterant agents, such as iodine, is not

advisable. This provokes reaction and extension and may prevent the subsequent use of mercurial preparations and antibiotics due to the contact complications present.

Avoid macerating tissue with excessively larger wet dressing than are required. Heat is the element required and dry heat is usually equally as effective as moist dressings. Poulticing must be done with caution if at all, in the treatment of furuncles.

Wet Dressings: The types of wet dressings are myriad and are best selected from those one is accustomed to using. Potassium permanganate makes a good soak but when used too long precipitation of the potassium permanganate will cause plugging of the follicles. The discoloration is also objectionable to many.

Saline magnesium sulphate and boric acid are old stand-bys and, when not used in too strong solutions, are effective.

Aluminum acetate or subacetate makes an excellent compress when used in either skimmed milk or water. We advocate using one teaspoonful to the pint of diluent, preferably cool or tepid, unless otherwise indicated. The reason for using skimmed milk is to get away from the rancid odor frequently encountered in butter fat or butyric acid. Sodium hyposulphite is a useful compress and soak especially when fungus infection is present.

When general immersion is required in cases of extensive involvement, the use of standard preparations such as boric acid, sodium bicarbonate, magnesium sulphate, sodium chloride, and various bland baths are recommended.

Tar baths are not advocated when secondary infection is present in the follicular and perifollicular areas because tar frequently will precipiate or aggravate a pustular eruption in those areas.

Potassium permanganate is recommended for bathing purposes for pustular conditions.

Reduction of the bacterial population, both surface and deep, is a necessity. The bactericidal index is sometimes dependent on the strength of the preparations used topically. Do not overtreat. Remember, the fundamental rule in treating dermatological conditions is never to treat acute dermatoses vigorously. It is preferable to undertreat rather than to overtreat these acute conditions.

The problem of treatment has of course been greatly simplified since the introduction of sulfonamides and antibiotics. The use of mild soap and water in acute cases, to cleanse thoroughly, is imperative. The removal of all detritus even to the use of curettage, if necessary, is encouraged. Cleansing the entire field frequently and adequately is one of the first rules to apply. Debridement is useful and time saving.

Kerotolytics are often required to soften, dissolve, and remove horny layers and crusts.

The alteration of the nutritional substance is recommended in the hands of the expert, especially is this true in the case of x-ray therapy. Therapeutic quartz and ultra violet therapy are excellent adjuncts to our therapeutic armamentarium, as these forms of active therapy have excellent bactericidal action. Here too one must not overtreat.

The correction of such physical factors as lymph and vascular stasis is fundamental, and reduction of any irritations of the pilosebaceous system is an absolute necessity.

We have already referred to the use of antiparasitics and one must be careful to strike a balance between skin health and germ destruction. Avoid therapeutic paradoxes. Too strong antiseptics are undesirable as they lower the patient's resistance and recuperative processes. The use of abortives or deterrents is often preferable to the use of so-called specifics. Remember the treatment of the individual rather than of the disease process is often the wiser course to follow.

The use of bactericidal agents is the common practice today; their worth is beyond question in these coccogenic dermatoses, both as topical applications and as

internal medications. The list of newer antibiotics is growing rapidly and one must find a suitable group and learn how to use them wisely. Some are best prescribed in ointment form and others are more effective in lotion form.

Ammoniated mercury and variants of this drug are the old reliable stand-bys. Lotions such as Alulotion (Wyeth) are effective in some cases. Proprietaries in combination with various drugs are recommended by many. We prefer to incorporate 2 or 3 per cent salicylic acid in most ointments unless contraindicated in cases where inflammatory reaction is present, as this drug is relatively innocuous and it helps remove the crusts and scales which are usually increased when the kernatinization cycle is stepped up, as is the rule in all inflammatory dermatoses.

Furacin has been used successfully in many of the inflammatory dermatoses. It is an antibiotic and is used as a compress and in ointment form. The danger of contact complications should be watched for, and the sensitization factor, at least in our hands, discourages its general usage. It is only fair to state that many of the sensitization faults of the drug have been corrected however.

Aureomycin ointment has been a great help in the treatment of coccogenic dermatoses. The tendency toward sensitization has been very slight in our experience.

Penicillin ointments are not used as they previously were because of the sensitization produced in many cases.

Bacitracin has been very useful to us in treating these eruptions. In fact, we frequently alternate it with aureomycin. There have been some contact dermatoses resulting from its usage, but one must remember it has been on the market longer and that fact may account for its more extensive usage.

Sulfonamides are not recommended for topical usage, as the complications associated with their local applications seem to render them too risky for general usage. Some specialists such as ophtholmologists still get satisfactory results from them however.

A much longer list could be given of topical applications which are very satisfactory in the treatment of coccogenic dermatosis but time will not permit such an inclusion.

The thing of importance is to get a drug that does the thing you want it to do in any preferable form, providing the complications are few, if any.

Internal medications: The medications to be used internally are so numerous since the advent of antibiotic therapy and sulfonamide therapy, that it has become a simple matter to select the one best tolerated by the patient and most effective in his particular case.

When one realizes how fortunate we are today with such a wonderful group of drugs to select from we wonder how we ever got some of the patients well in the not too distant past.

Penicillin is, of course, one of the drugs of choice and may be used in liquid form or tablet form and is effective in both methods of administration. The parenteral and intravenous forms are more rapid in action however. We cannot speak for the lozenge form of penicillin as our experience in that form is very limited.

The complications are not too numerous and we have found urticaria, angioneurotic edema, and erythema multiforme are the principal complications. Occasionally, Herxheimer's reaction is encountered when penicillin is used in antisyphilitic therapy.

Streptomycin is of definite value in certain infections, particularly in bacillary and virus infections, but we have had to resort to its use in but a few cases of coccogenic dermatoses.

Aureomycin has been very effective in treating coccogenic infections as well as virus infections, when given internally both parenterally and in capsule form. The difficulty is the cost of the medication which is entirely too high for general usage. The complications are few since the nausea factor has been corrected. Pruritis, especially perianal and perivulvar, which is caused in

some cases, requires caution in the usage of this drug.

Chloromycetin is also very effective and as an internal medication compares very favorably with aureomycin.

Sulfonamides are still used internally in combating coccogenic dermatoses. We prefer sulfadiazine in tablet form 7.7 grain tablets, one to two, four times a day, with sodium bicarbonate or some alkali, which has been of great value to us when penicillin was not tolerated and an inexpensive substitute was needed. It is especially valuable in the treatment of coccogenic infections, when there is an associated mycotic infection present.

Medical care is replete with the value of vaccines, both autogenous and stock vaccines, in the treatment of coccogenic infections.

The subject of synergism is not new but it is rarely seen. The use of antibiotics which show such synergistic properties is of recent interest. Synergism in drugs represents a phenemenon in which two or more drugs potentiate each other's therapeutic action.

According to the works of Eagle and Fleischman[1] true synergism is produced when bacitracin and penicillin are given together. True synergism is present when an amount of each drug is given which is considerably smaller than the curative dose of either; yet the mixture is itself curative.

We have found that penicillin given parenterally and sulfonamides when given orally in the form of sulfadiazine show a tendency to work synergistically.

Correction of local resistance reducing factors is of utmost importance. The avoidance of follicular irritation caused by the rubbing of dressings and the reaction to adhesive tape, especially in hot weather, is an important one. The correction of occupational and postural injuries should be taken care of. The use of supports and mechanical aides, such as supportive bandages and fixed dressings, requires study and application as indicated. Rest in bed is of paramount importance. Someone has said that rest in bed helps all illness except morphine poisoning and there is some merit to that assertion. Protect and support the necessary areas, and the individual will be helped. The correction of these local resistance reducing factors is very necessary.

Correction of systemic factors: Rest as a therapeutic aid is known to all, but this rest must be mental as well as physical. Freedom from worry is a most necessary measure to be sought for, especially by the sick patient.

The care of the general problems of health, such as regulation of the bowels, correction of dietary problem with special attention to protein consumption, is desirable. A reduction of carbohydrate intake is stressed for some cases. The vitamin requirement is an important consideration. When indicated, blood count and urinalysis are suggested and medications recommended for low blood counts. In some cases basal metabolism is advised to detect and correct faulty factors there.

The increase of resistance by use of vaccines has already been touched upon and should merit serious consideration in selected recalcitrant cases.

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MALIGNANT LESIONS OF THE SKIN

JAMES W. TEDDER, M. D.
NEW ORLEANS

Malignant lesions of the skin are very numerous; and, in addition, they are visible and readily accessible to, and for, treatment. These lesions occur more frequently in men than in women; also, they occur more often in people who are exposed to the elements such as sun, wind, and salt water. The exposure factor is proved by the fact that the incidence of skin cancer in Navy men as compared with a comparable group of men in civilian life, has been found to be eight times as high. Skin cancer is found in all ages, but most frequently in those past the age of fifty.

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The types of malignancy are very numerous, and not infrequently may change from a fairly benign type to a more malignant growth.

The most commonly encountered lesions of the skin are the basal cell epitheliomas that spread locally, and the squamous type cell which may metastasize to adjacent glands. A third type of common epithelioma is one of the transition between the two previously mentioned types and is classified as a "mixed cell" epithelioma. One group of skin lesions that comes in for considerable discussion is the "pigmented nevi", which may range in size and color from that of a pinpoint to several inches in diameter.

It has been my policy to remove pigmented lesions only when they are subject to trauma, when they show evidence of increasing in size, and when they are subject to removal for cosmetic reasons. The larger percentage of the epitheliomas occur on the exposed parts of the body and are usually just watched (by the victim). Many bad lesions have just been watched (by the patient) for a period of many months, or even over a period of many years, and by the time the patient finally decides to seek medical assistance, the lesions have progressed beyond such help.

It is advisable, in any general physical examination, to look for such lesions and explain their nature to the patient, especially in the older age group. The patient should be encouraged to have these lesions treated when they are first discovered. The earlier the reatment, the more successful the final results!

There are various methods of treatment in use. The majority of these treatments give satisfactory results in the proper hands. Surgery, x-ray, radium, radon implants and cautery are all strongly advocated by their particular devotees. I, personally, have found that a combination of surgery, cautery and x-ray gives a 95 per cent satisfactory result, if the lesion is seen before extensive spread or metastasis occurs.

It is strongly urged that one should always take a biopsy of the suspected lesion when in doubt, or even routinely, because it is impossible—in many cases—to make a clinical diagnosis, and there is also the additional necessity for confirming the proof in the event the treatment is ever questioned.

A biopsy, which I routinely do by the punch method, is very satisfactory and results in no scar formation if the lesion does not prove to be malignant. The punch that I prefer to use is ½ millimeter in diameter. No anesthesia is necessary, and the punch is applied with firm pressure and a rotating motion, being sure to obtain full skin thickness for the biopsy.

As I have mentioned previously, if no malignancy exists, the lesion heals with a minimal scar that is not visible except upon close examination. This method is especially desirable where lesions appear on the face.

In those cases in which the lesions are malignant, or one is clinically convinced that their removal is necessary, it may be done at the time of biopsy. It has never been proved that biopsy has caused a nonmalignant lesion to become malignant or to metastasize.

Very frequently, when I am reasonably sure of the nature of the lesion, in addition to the removal and cauterization, I immediately follow this treatment with x-ray therapy. This therapy is usually divided into three sessions, consisting of from three to five erythema doses, with or without one filter, the total dosage amounting to from 1,200 to 4,800 "R" units. The use of x-ray over cartilage is to be avoided as it may result in the absorption of cartilage structure.

Where x-ray or radium is not readily available, I feel that they should be sought out by the physician responsible for the successful treatment of the case. A satisfactory result is difficult to obtain by excision alone; however, when this method is used, a margin of normal appearing tissue must also be excised, plus a thorough subcutaneous removal. These lesions may be closed by primary sutures and close observation for a period of from six months to five years followed. This, I consider inadequate therapy.

The so called "premalignant" lesion, such as cornu cutaneum, is to be respected. If, after local medication, it does not appear to respond, then it should be removed. Here, again microscopic examination is a most helpful aid. Areas of chronic inflammation, lupus vulgaris, lupus erythematosus, roentgen dermatitis, chronic ulceration, and old burn scars are precancerous lesions and should be regarded as such.

There are approximately a half million new cases of cancer each year, and over 150,000 deaths each year have been directly attributed to cancer. A study has indicated that the patient is making progress in reducing the period of delay in the diagnosing and treatment of lesions although the physician is not. The principle available means of reducing morbidity and mortality due to cancer are early diagnosis and treatment.

In conclusion, I would like to again remind you, that it is the important responsibility of the general practitioner to recognize, and treat, the early malignancies of the skin; and, never to let your patient "watch a lesion grow". Always give the patient the benefit of a microscopic examination and a biopsy. Early and adequate therapy is essential, regardless of the method used. The first treatment is the most important because recurrences are far more difficult to treat than is the original lesion!

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DISCUSSION

Dr. Richard W. Vincent (New Orleans): As Dr. Tedder stated, it is most important that an early diagnosis be made and that thorough, complete treatment be given by the one who first treats the carcinoma.

The choice of treatment, whether surgery or irradiation therapy, is not so important as the quality of the treatment. If surgery is used, adequate excision must be done. If irradiation is the method of choice, adequate treatment must be given. If the carcnioma is not cured by irradiation it is unwise to repeat the treatment and surgery should be elected.

Dr. James W. Tedder (New Orleans): I think Dr. Howles' paper was a very comprehensive review of coccogenic infections. I would like to add that in the use of wet packs, if you send a patient to the hospital and prescribe the application of wet packs, nine times out of ten when you get there they have a wet pack on, but it is covered with oiled silk, or some nonporous material. The whole idea, as I get it, in the application of wet packs is that it is osmosis you want. The pack is composed of a dozen thicknesses or so of gauze and you keep them saturated with the solution. As the solution evaporates, it draws out the inflammation and helps relieve the infection deep in the tissue. One of my favorites when a pack solution is required is the old Darier's solution. I don't like potassium permanganate because of the discoloration. I like copper sulfate. It will do anything that aluminum acetate or potassium permanganate will do, plus the fact that it smells good.

Dr. James K. Howles, (New Orleans): Dr. Tedder, there is one thing I didn't mention—I didn't have the time. I find it easy to prevent difficulty with the wet pack by inserting a Dakin tube in the pack; insert it into the dressing, using several thicknesses of the gauze and, of course, a Dakin tube is tied off at the one end and just inject some of the material into the tube. You keep the deep areas of the dressing wet. That is very difficult unless we have cooperative personnel on our services. One of our difficulties is that we don't have private hospitals that cater to dermatological cases. We have a difficult time getting things done right. I find that this procedure helps a lot in the osmotic process.

Dr. M. T. Van Studdiford (New Orleans): I think Dr. Howles amplified very beautifully the significance of the face infections, especially impetigo of the beard and the treatment thereof.

Dr. L. D. McLean (New Orleans): I would like to make one remark in regard to biopsy. We commonly think of the biopsy as being an aid in proving a lesion malignant, so that it may be treated thoroughly enough to completely destroy all carcinoma cells. Sometimes, however, a biopsy may help a great deal in the opposite direction, by proving that a malignant-appearing lesion is a benign growth. You may remember a case that we had in conference at Charity Hospital in New Orleans, where a lesion was suspected of being a malignant one. It was recommended that a biopsy be performed but instead the operating surgeon disregarded this recommendation and radical surgery was performed including wide excision, skin strips removed between the lesion, and the regional lymph nodes and inguinal lymph node dissection performed. The sections from the malignant tumor proved it to be a perfectly benign hemangioma. I think a biopsy is very important.

THE PATHOLOGIC CHANGES IN THE HEMATOPOIETIC SYSTEM FOLLOWING ANTILYMPHOMATOUS DRUGS

PHILIP PIZZOLATO, M. D.
NEW ORLEANS

NITROGEN MUSTARD

Many studies have been made on the effect of nitrogen mustard on the hematopoietic organs of animals and man. Kindred[1] and Karnofsky and Smith[2] have made comprehensive studies on animals, and have come to the same conclusions. In the lymphoid organs the tissue shrinks in volume because of the destruction of the lymphocytes, the inhibition of mitosis, and the migration into the blood stream. The reticulum cells are not injured directly by the mustard. These cells ingest and digest the degenerated lymphocytes. Regeneration of the lymphoid tissue begins on the third day after injection and is believed to arise from small lymphocytes which escaped the toxic action of the mustard.

Spitz[3] in a study of the post-mortem tissues of 57 patients with a variety of lymphomas and other malignant tumors observed the outstanding cytological effects on Hodgkin's disease and in reticulum cell sarcoma. These changes were most conspicuous in lymph nodes about seven days after therapy and consisted of "cytoplasmic ballooning" and nuclear swelling and degeneration. An increase of fat was noted in these cells. Other cells showed an increased pleomorphism. Mitoses were not numerous and lymphocytes were decreased. The bone marrow revealed different degrees of depression; the granulocytes being most seriously affected. In severe cases there was complete aplasia; the fat cells were sharply defined and appeared to be floating in an edematous matrix. In extreme cases the fat was replaced by a myxomatous gelatinous substance.

Cornell and Blauw[4] observed intense phagocytosis of lymphocytic nuclei and their fragments by lymphocytes five days after a course of mustard therapy in a case of Hodgkin's disease. Alpert and Peterson[5] reported necrosis of the reticulum cells, giant cells, and eosinophils with intact lymphocytes in lymph nodes of two patients with Hodgkin's disease after three days and two weeks respectively of nitrogen mustard therapy.

In our series we have studied the effect of nitrogen mustard on 38 patients; 9 had malignant lymphomas, 7 of whom died. Post-mortem examination was performed on 6. There were 29 patients with nonlymphomatous neoplasms, of whom 13 died. Our post-mortem findings resembled those of Spitz.[3] During life, however, many patients had serial marrow studies before and after treatment. It is to be noted that a depression of the total nucleated elements[6] in all cases was encountered; however, the differential count was essentially unchanged. (Table 1)

TABLE 1

BONE MARROW STUDY
TOTAL NUCLEATED ELEMENTS IN THOUSANDS

Diagnosis	Before Therapy	After Therapy
Hodgkin's disease	80.5	31.0
Hodgkin's disease	50.0	6.4
Fibromyxosarcoma	111.8	30.0
Carcinoma of lung	76.0	4.2
Carcinoma of lung	57.3	15.0

URETHANE

Most observations on urethane deal with a quantitative change in the lymphoma cell rather than a cytologic alteration. Murphy and Sturm[7] using rats with lymphatic leukemia observed a decrease of lymphocytes and a slight decrease in the size of the lymph nodes, thymus, and spleen. Kirschbaum and Lu[8] found that urethane produced in mice myelogenous leukemia, a drop in the white cell count, and the appearance of

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From the Clinical Laboratory, Veterans Administration Hospital; Department of Pathology, Louisiana State University School of Medicine and Charity Hospital, New Orleans, Louisiana.

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many mature cells in the bone marrow. Mitotic figures in the marrow myeloid cells also decreased.

Berman and Axelrod[9] found that the lymph node in human leukemic reticuloendotheliosis after urethane became smaller and softer with numerous areas of irregular replacement of lymphoid tissue by a loose edematous collagenous connective tissue. There were also capillary dilatations, pyknosis of nuclei, karyorrhexis, and infiltration with hemosiderin-laden macrophages. In a case of subacute leukemia they found that the lymph nodes showed nuclear degeneration among the leukemic cells. They also found that urethane causes a fall in the absolute number of both lymphocytes and granulocytes in the peripheral blood. The effect was greater on the more undifferentiated cell. The bone marrow may undergo temporary or permanent hypoplasia. Megakaryocytogenesis persisted longer than erythrocytogenesis or leukogenesis and appeared to recover earliest after urethane injury.

In 1 of the 2 cases reported by Alwall[10] urethane produced in myeloma a decrease of marrow myeloma cells from 33 per cent to 20 per cent in one and a half months, and after three and a half months of treatment, no myeloma cells were found. His other case did not respond to urethane but did respond to stilbamidine.

Loge and Rundles[11] in their study of 4 cases of urethane treated myeloma observed a disappearance of immature granulocytes and normoblasts from the peripheral blood and a change in the myeloma cells characterized by an increase in size, pyknosis, and a greater eccentricity of the nuclei, and the tendency for the cells to persist in small clumps.

In our study, we used urethane on 4 patients. One of these who had chronic myelogenous leukemia did not respond to the drug but did respond to deep x-ray therapy. The other patients who had multiple myeloma did not respond to the treatment and subsequently died. Autopsy did not reveal any findings attributable to the urethane.

FOLIC ACID ANTAGONISTS

Philips and co-workers[12] noted that the marrow of rats receiving aminopteroylglutamic acid changed from a greyish red gelatinous marrow to a dark liquid type. The sinus and capillary network of the marrow became prominent due to depletion of cells. Eosinophils, megakaryocytes, and normoblasts were the remaining cells. There was a corresponding granulocytopenia, lymphocytopenia, and reticulocytopenia in the peripheral blood. With the exception of lymphocytic necrosis in the thymic cortex, changes in the lymphoid tissue were not encountered. Dameshek[13] and Farber[14] in a preliminary report did not present their autopsy finding in their study of the effect of the folic acid antagonistic drugs on the acute leukemia. We studied the post-mortem material from 5 cases of acute leukemia receiving the drugs. Of these a hypoplastic marrow was the only finding attributable to aminopterin.

STILBAMIDINE

Snapper and co-workers[15, 16] observed patients with multiple myeloma treated with stilbamidine developed basophilic inclusions in the myeloma cells when stained with Wright stain. These inclusions stain as large red globules with supravital stain, and with Unna stain bright red granules are seen in the cytoplasm. These bodies contain ribose nucleic acid.

Similar findings were found by Proff and co-workers.[17] We have been able to find the basophilic inclusion bodies in the plasma cells following stilbamidine in one case of multiple myeloma. This patient subsequently died and no alterations of the hematopoietic system due to this drug were observed.

CONCLUSIONS

The action of nitrogen mustard, urethane, aminopterin and stilbamidine on the hematopoietic system of animals and man is discussed. These drugs are cytotoxic to a varying degree to granulocytic, erythroid, lymphoid, and megakaryocytic elements. Although many claim to get favorable response to these agents on the various lymphomata, our experience has been very discouraging.

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DISCUSSION

Question—Dr. Russell Lowell Holman (New Orleans):

I think Dr. Pizzolato is to be congratulated on his meticulous study of the bone marrow. I would like to ask whether any of these actions you have mentioned have been shown in vitro or in tissue culture to have any effect on the cells?

Answer—Dr. Pizzolato:

I think Dr. Pomerat from Texas is doing some work, and I haven't been able to get him to make any definite commitments. I haven't seen anything written by any of the others.

Question—Dr. Spears Randall (New Orleans):

I would like to ask this: We have observed that nitrogen mustard in neoplastic conditions other than the lymphomas produces rather startling clinical improvement. This is not in selected cases, because they are unpredictable improvements; but, in cases, particularly of carcinoma of the lung. Many of these patients are taken from death's door very dramatically and walk out of the hospital feeling fine. A few of those have come to further study at autopsy, and, as in the hemopoietic system, there is not much change in the cell itself. Roentgenologically how many of these show striking decrease in the size of the tumors? I wonder if you have seen that?

Answer—Dr. Pizzolato:

Sad to say, we cannot agree with the work of others, because in our cases of bronchogenic carcinoma, nearly all died, or were at least in the process of dying. In very few of the cases could we say there is a nice example of response. I think some of these are treated rather early. It is very disconcerting. We would like to try something else, if we had something better, on these bronchogenics. They all died, and they died, it seemed, more rapidly than would have been the case if they had been left alone.

Question—Dr. Randall:

I do not say they do not die. They do, if they have a carcinoma. But there is a very dramatic improvement in a lot of them. We have some at the hospital that have temporary improvement, even if they do die.

Answer—Dr. Pizzolato:

They may respond clinically. But, as I said before, they get a course of mustard, and before they get a subsequent course of mustard, we always check the bone marrow. So, I see these patients clinically; and shortly after that, on the second or third marrow study, I find I could do one in the morgue. So, as I said, at least maybe some of the clinicians might feel a little pleased with the results. As far as the pathologist is concerned, the results are very disconcerting.

Question—Dr. Randall:

But clinically speaking, some of them do improve, I think. With the availability of the nitrogen mustard in general, I think the treatment will show some gratification. When your patients come in cyanotic, for all practical purposes dead, and you start them on nitrogen mustard and in a matter of a week they are able to go back home free of the symptoms they came in with, it is very dramatic. Maybe they like our hospital, because they stay with us.

Answer—Dr. M. T. Green (Ruston):

I would like to confirm the doctor's statement as to palliation. True, mustard offers nothing curative and is not indicated from the laboratory point of view. We who care for these patients in private practice concentrate on the patient's sense of well being since we are unable to offer them any relief that is curative. Many of these patients are most grateful for a few doses of mustard. They are made comfortable and are able to go home and enjoy a few days of comfort.

If we can give these unfortunate people a few days of palliation, we have done something for them.

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THERE IS NO NATIONAL SHORTAGE OF PHYSICIANS

To some extent in the last several years, and to a considerable degree in recent months, there has been comment about a supposed national shortage of physicians. A sharp degree of public interest has been raised on this point in a recent article in *Collier's* magazine by Albert Q. Maisel. In substance, the article in *Collier's* stated that there was an alarming shortage of doctors because of action on the part of leaders of the A. M. A. The well meaning but presumably misguided attitude of these leaders caused them to thwart legislation designed to provide federal aid for medical education.

These two problems, namely, that of availability of doctors and that of assistance to medical schools, are separate. Discussing the first, the article referred to above made such statements as this: In the action of the leaders of the A. M. A., "Because of what they have done and what they have prevented from being done, civilian atomic-bomb victims may perish untended. Mothers, lacking a physician's help, may die in childbirth under the ministrations of incompetent midwives. Plagues and epidemics, which we all thought banished forever, may return to claim millions of victims." Editorial comment in the magazine said that the stand of the A. M. A. was wrong; their attitude was narrow, and their case was weak. It stated further that few would bother to deny the present shortage of medical personnel or to deny the actual potential danger of such a shortage. Reviewing the matter of physician shortage, Maisel stated that in 1910 medical schools were producing one new doctor for every 20,000 of the population; in 1950, one new doctor was graduated for every 27,000 population. To those who are familiar with the course of medical education and the extent to which standards of education and the training of physicians have been improved under the assistance of the A. M. A., the answer is obvious. In 1910, physicians were being graduated from weak medical schools and diploma mills. These diploma mills are nonexistent now. Accordingly, the significance of the term "physician" with respect to training and ability differs markedly today from what it was in 1910. The number of physicians per 100,000 population in 1909 was 149, and in 1949, it was 137. Nevertheless, the United States has today the largest number of physicians per 100,000 population of any nation in the world except Palestine. In Jewish Palestine the doctors per 100,000 population are 382. The next below the United States is Great Britain with 114. In addition to our

having the largest number of doctors in proportion to population of any large country, the number of graduates of Class A medical schools has had an upward trend since 1910. In 1950, our physician output amounted to 5,553. There is indication, as is shown by the study of the tables in Dickinson's analysis of this situation, that there is every likelihood that the number of medical school graduates will rise to 7000 in about ten or twelve years when all the expansion that is now under way is in full effect. A similar study has shown that the number of physicians has increased more rapidly than the population since approximately 1929.

In the years which are being considered in the so-called problem of the doctor shortage, the efficiency of the average doctor has increased from one third to one half. The factors which have contributed to this are better training, decreased amount of time spent in transportation, increased efficiency of the doctors' effort which was provided by technological specialists in the various fields adjuvant to medicine. The combined effect of these various factors means that fewer doctors can take care of more patients adequately. In World War II there was given adequate proof of the statement just made. Approximately 60 per cent of the doctors remained in the civilian population and took very good care of the 90 per cent of the population which also remained civilian. It is well known that the medical service rendered in this fashion was adequate as there was a very fine civilian health record during World War II. In further support of the character and quality of medical service rendered by the physicians as a group may be cited the increase in life expectancy, which in 1910 was 51 years, and in 1940, 64 years, and in 1950, 69 years, an increase of 18 years during the life span of most physicians now practicing.

There is, then, no doctor shortage so far as regards numbers. As regards medical service there may be an inadequacy of opportunity in certain localities. This fact is based partly on facilities and partly on economic considerations. These deficiencies will have to be met on their own grounds unless we as a nation were to retrogress rapidly into a welfare, and ultimately, a police state in which doctors could be assigned to localities at the will of the dictator. As regards the action of the A. M. A. in thwarting the direct subsidy of medical schools by the federal government, this has been discussed in these columns on previous occasions. It is perfectly obvious that the action of leaders of the A. M. A. has the support of organized medicine, and that the profession stands against the subsidy of medical schools by the Federal government.

It should be clear that if such a subsidy were put into operation at the present time it would have very little effect on the available doctor supply until some time in 1957, at the very earliest. It would take at least six, and probably more years to materially increase medical school facilities, and at the same time to appreciably increase the number of graduates in those schools. It is perfectly obvious that the threat of pestilence envisaged by the author of the *Collier's* article could not be minimized in the near future by the efforts of medical students who have not yet started to school.

The position, therefore, of the physician in organized medicine is clear and it is one of which the profession could well be proud. The character and quality of medical service is improving. It is the best there is. It is becoming increasingly more available. We have the greatest number of physicians of any large country. The problem that confronts us is one of distribution. The health record of the nation attests to the correctness of these points of view.

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The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

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Secretary—Dr. Charles Anderson, 1580 Line Avenue, Shreveport

CALCASIEU PARISH

President—Dr. C. V. Hatchette, Lake Charles
Secretary-Treasurer—Dr. T. A. Dees, 202 Foster Street, Lake Charles

CLAIBORNE PARISH

President—Dr. Martin Luther Forcht, Jr., Haynesville
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CONCORDIA-CATAHOULA BI-PARISH

Inactive

DE SOTO PARISH

President—Dr. L. S. Huckabay, Coushatta
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EAST BATON ROUGE PARISH

President—Dr. Daniel J. Fourrier, Baton Rouge
Secretary-Treasurer—Dr. John Ray Powers, 3170 Florida Avenue, Baton Rouge

EAST AND WEST FELICIANA BI-PARISH

President—Dr. Eugene M. Robards, Jackson
Secretary-Treasurer—Dr. Enoch McLain Toler, Clinton

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President—Dr. R. E. Dupre, Ville Platte
Secretary-Treasurer—Dr. R. B. Thompson, Ville Platte

FRANKLIN PARISH

President—Dr. Samuel Carriere, Winnsboro
Secretary-Treasurer — Dr. Frank R. Preston, Winnsboro

IBERIA PARISH

President—Dr. L. M. Villien, Jeanerette
Secretary-Treasurer—Dr. G. R. Guiberteau, Jeanerette

IBERVILLE PARISH

President—Dr. F. O. Tomeny, White Castle
Secretary-Treasurer—Dr. E. C. Melton, Plaquemine

JACKSON-LINCOLN-UNION

President—Dr. John A. Thomas, Ruston
Secretary-Treasurer—Dr. J. L. Smith, Jr., Green Clinic, Ruston

JEFFERSON DAVIS PARISH

President—Dr. R. F. Miller, Jennings
Secretary-Treasurer—Dr. J. R. Romero, Welsh

LAFAYETTE PARISH

President—Dr. Don B. Williams, Lafayette
Secretary-Treasurer—Dr. R. H. Robinson, 314 W. St. Mary Street, Lafayette

LAFOURCHE PARISH

President—Dr. Joseph W. Brown, Lockport
Secretary-Treasurer—Dr. Richard E. Robichaux, Raceland.

MOREHOUSE PARISH

President—Dr. Otis L. Tugwell, Jr., Bastrop
Secretary-Treasurer — Dr. Bernard Soto, Mer Rouge

NATCHITOCHES PARISH

President—Dr. W. W. Knipmeyer, Natchitoches

ORLEANS PARISH

President—Dr. Boni J. DeLaureal, 411 Medical Arts Bldg., New Orleans
Secretary—Dr. Charles B. Odom, 1430 Tulane Avenue, New Orleans.

OUACHITA PARISH

President—Dr. H. E. Guerriero, Monroe
Secretary-Treasurer—Dr. C. T. Yancey, Monroe

PLAQUEMINES PARISH
Inactive

POINTE COUPEE PARISH
President—Dr. F. F. Rougon, New Roads
Secretary-Treasurer—Dr. Anna Plauche, Morganza

RAPIDES PARISH
President—Dr. Henry C. Gahagan, Alexandria
Secretary-Treasurer—Dr. A. E. Hensel, Alexandria

RED RIVER PARISH
Inactive

RICHLAND PARISH
Inactive

SABINE PARISH
President—Dr. L. H. Murdock, Zwolle
Secretary-Treasurer—Dr. Rufus H. Craig, Many

ST. LANDRY PARISH
President—Dr. J. T. Thompson, Eunice
Secretary-Treasurer—Dr. Webster LaHaye, Eunice

ST. MARTIN PARISH
President—Dr. L. A. Morrogh, Breaux Bridge
Secretary-Treasurer—Dr. Robert L. Morrow, Arnaudville

ST. MARY PARISH
President—Dr. F. H. Metz, Morgan City
Secretary-Treasurer—Dr. Hilton J. Brown, Franklin

ST. TAMMANY PARISH
President—Dr. Henry T. Cook, Slidell
Secretary-Treasurer—Dr. M. J. Duplantis, 322 Columbia St, Covington

TANGIPAHOA PARISH
President—Dr. Alexander J. Feder, Box 130, Hammond
Secretary-Treasurer—Dr. Ephraim Lubritz, Amite

TERREBONNE PARISH
President—Dr. Clark Collins, Houma
Secretary-Treasurer—Dr. Buford J. Autin, Houma

TRI-PARISH (EAST AND WEST CARROLL, MADISON AND TENSAS)
Inactive

VERMILION PARISH
President—Dr. Thomas Latiolais, Sr., Kaplan
Secretary-Treasurer—Dr. Ed. J. LeBlanc, Erath

VERNON PARISH
President—Dr. Edwin H. Byrd, Leesville
Secretary-Treasurer—Dr. T. J. Lawrence, Leesville

WASHINGTON PARISH
President—Dr. Paul D. Gard, Bogalusa
Secretary-Treasurer—Dr. C. W. Crain, Bogalusa

WEBSTER PARISH
President—Dr. C. S. Sentell, Minden
Secretary-Treasurer—Dr. W. C. Gray, Springhill

SECOND DISTRICT
President—Dr. Joseph H. Massony, Westwego
Secretary-Treasurer—Dr. Frederick A. Wild, Jr., Destrehan

THIRD DISTRICT
President—Dr. M. A. Young, Abbeville
Secretary-Treasurer—Dr. E. P. Breaux, Lafayette

FOURTH DISTRICT
President—Dr. J. E. Holoubek, 207 Phys. & Surg. Bldg., Srheveport
Secretary-Treasurer—Dr. O. C. Thomas, 265 Robinson, Shreveport

FIFTH DISTRICT
President—Dr. Carroll Summer, Monroe
Secretary-Treasurer—Dr. Hayden Cutler, Monroe

SIXTH DISTRICT
President—Dr. J. D. Thames, Hammond
Secretary-Treasurer—Dr. B. B. Lane, Zachary

SEVENTH DISTRICT
President—Dr. A. H. LaFargue, Sulphur
Secretary-Treasurer—Dr. J. B. Hodge, Jr., Sulphur

EIGHTH DISTRICT
President—Dr. B. H. Texada, Alexandria
Secretary-Treasurer—Dr. A. E. Hensel, Alexandria

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LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Third Thursday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

E. & W. FELICIANA BI-PARISH MEDICAL SOCIETY

A dinner, scientific and business meeting was held by the E. & W. Feliciana Bi-Parish Medical Society in Clinton on December 20. Dr. C. Grenes Cole, Secretary of the State Society presented a paper on socialized medicine and compulsory medical and surgical insurance after which a resolution was passed requesting citizens of Louisiana to write or wire their U. S. senators and congressmen asking them to vote against proposed legislation for this type of medical practice. Resolution re-

questing publication of Dr. Cole's paper in the New Orleans Medical and Surgical Jaurnal and in the Journal of the American Medical Association was also adopted.

Dr. C. Grenes Cole and Dr. Edward O. Munson, Jr., were elected to honorary membership in the society and the following doctors were elected to serve as officers during 1951: Dr. E. M. Robards, President; Dr. W. J. Roberts, Vice-President; Dr. E. M. Toler, Secretary-Treasurer.

The next meeting of the organization will be held in Jackson on the first Wednesday in March.

NEWS ITEMS

Dr. Lucien A. LeDoux, of New Orleans, was recently elected to fellowship in the American Association of Abdominal Surgeons, Gynecologists and Obstetricians.

Dr. Ambrose H. Storck has been appointed to the Advisory Board of the International Abstracts of Surgery.

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY

At a special meeting of the American Board of Obstetrics and Gynecology, held in Pittsburgh, Pennsylvania, December 14, the following changes in the regulations of the Board were unanimously adopted:

1. That physicians otherwise qualified, who were graduated before January 1, 1949 and whose required training was in obstetrics or gynecology alone, and who have confined their practice to obstetrics or gynecology for at least five (5) years immediately prior to application be accepted for examination as candidates for certification in either obstetrics or gynecology. In all other respects requirements for eligibility remain the same for those physicians graduated since 1939. Bilateral training is required as published in the Bulletin of the Board.

2. Applicants who have been certified by one of the other American Specialty Examining Boards will not be eligible for certification by this Board until they have relinquished the certificate previously conferred.

3. Since the vast majority of obstetrical and gynecological cases are non-operative the Board requires adequate training in basic sciences, infertility, endocrinology, oncology, irradiation therapy, psychosomatic medicine, electrotherapy, and other non-operative methods of diagnosis and treatment as well as training in major operative procedures.

AMERICAN BOARD OF CLINICAL CHEMISTRY, INC.

A certifying board, the American Board of Clinical Chemistry, Inc., has been established by representatives from the American Chemical Society, Inc., American Institute of Chemists, Inc., and the American Society of Biological Chemists, Inc. It is the purpose of this Board to encourage the study, improve the practice, elevate and establish standards and advance the science of that specialized branch of chemistry entitled "clinical chemistry"; to grant and issue certificates or other recognition of special knowledge in clinical chemistry to voluntary applicants who in accordance with the by-laws, rules and regulations of the Board have established their fitness and competence; to prepare and furnish in the public interest a registry of individuals with specialized knowledge in clinical chemistry who have from time to time been granted certificates by the Board. The Board is analagous in purpose, function and organization to the various medical specialty boards which have served for many years to establish standards and qualifications for persons wishing to practice in a specialized field in medicine.

Further information may be obtained from the office of the Secretary-Treasurer, Dr. Jos. W. E. Harrisson, 1921 Walnut Street, Philadelphia 3, Pennsylvania. A fee of $1.00 must accompany the application request, to cover clerical and mailing costs. All requests should give the name of the applicant in the manner it is to be recorded, as application forms will be so issued.

RADIOLOGIC DEFENSE

The American College of Radiology has instituted a course of instruction in Radiologic Defense underlining the responsibility of radiologists in atomic attacks. Services of local radiologists should therefore be solicited in the development of local civil defense programs.

AMERICAN COLLEGE OF ALLERGISTS

The seventh annual meeting of the American College of Allergists will be held at the Edgewater Beach Hotel, Chicago, February 12-14. There will be a post-graduate instructional course on the three days just preceding the annual meeting. This course will be an extremely practical one, designed for any physician who wants to learn the basic principles of diagnosis and treatment of allergic individuals and technics that are useful in the management of these patients. A fee of $35.00 will be charged for the three-day course. For further information write Dr. Fred Wittich, Secretary-Treasurer, LaSalle Medical Building, Minneapolis, Minnesota.

SOUTHWEST ALLERGY FORUM

The Southwest Allergy Forum will hold a meeting at the Plaza Hotel in San Antonio, Texas, April 8-10. All doctors interested in allergy are extended an invitation to attend this meeting.

NEUROPSYCHIATRIC MEETING

The Third Annual Neuropsychiatric Meeting will be held at the Veterans Administration Hos-

pital, North Little Rock, Arkansas March 1-2. A number of nationally known figures in neuropsychiatry and related fields have accepted invitation to participate in this meeting and in addition there will be many professional exhibits and clinical demonstrations relative to the hospital and its varied diagnostic and therapeutic activities. A special conference for nurses will be held on February 28 and psychologists and social workers will have sessions at regular intervals during the meeting. There is no registration fee and attendance of all interested professional personnel will be welcomed subject only to limitation of seating facilities. For further information communicate with Dr. Ewin S. Chappell, Director of Professional Education, Veterans Administration Hospital, North Little Rock, Arkansas.

NATIONAL GASTROENTEROLOGICAL ASSOCIATION CONTEST

One hundred dollars and a Certificate of Merit will be awarded by the National Gastroenterological Association for the best unpublished contribution on gastroenterology or allied subjects. In addition certificates will be awarded those physicians whose contributions are deemed worthy. Contestants residing in the United States must be members of the American Medical Association. Those residing in foreign countries must be members of a similar organization in their own country. The winning contribution will be selected by a board of impartial judges and the award will be made at the annual convention banquet of the National Gastroenterological Association in September. The Association reserves the exclusive right of publishing the winning contribution and those receiving Certificates of Merit, in its official publication *The Review of Gastroenterology.* All entries for the 1951 prize should be limited to 5,000 words, be typewritten in English, prepared in manuscript form, submitted in five copies accompanied by an entry letter, and must be received not later than June 1, 1951. Entries should be addressed to the National Gastroenterological Association, 1819 Broadway, New York 23, N. Y.

TEACHING SEMINAR IN PROCTOLOGY

The International Academy of Proctology will present a teaching seminar in New York City, April 7. Registration will be limited in number and open to licensed physicians who are members of the American Medical Association, state and county medical societies and graduates of an approved medical school. Admission to the seminar will be by card only, however preference in registration will be given to those affiliated with the International Academy of Proctology. Further information may be obtained by communicating with Dr. William Lieberman, Chairman, Seminar Committee, International Academy of Proctology, 1819 Broadway, New York 23, N. Y.

THE AMERICAN CONGRESS OF PHYSICAL MEDICINE

The twenty-ninth annual scientific and clinical session of the American Congress of Physical Medicine will be held in Denver, September 4-8. All sessions will be open to physicians and other professional personnel and in addition to the scientific sessions annual instruction seminars will be held September 4-7. These seminars will be offered in two groups. One set of ten lectures will consist of basic subjects and attendance will be limited to physicans. Another set of ten lectures will be more general in character and will be open to physicians as well as to therapists, who are registered with the American Registry of Physical Therapists or the American Occupations Therapy Association. Full information may be obtained by writing to the American Congress of Physical Medicine, 30 North Michigan Avenue, Chicago 2, Illinois.

WOMAN'S AUXILIARY TO THE LOUISIANA STATE MEDICAL SOCIETY

EAST BATON ROUGE PARISH

Mrs. DeWitt Milam of Monroe, President of the Auxiliary to the State Medical Society and Mrs. Joseph Heard of Shreveport, First Vice-President of the State Auxiliary, were special guests at a meeting of the Auxiliary to the East Baton Rouge Medical Society held in December.

A tea was also recently given by the East Baton Rouge Auxiliary in honor of members of the Louisiana Nurses Association in attendance at their annual convention held in Baton Rouge.

LAFAYETTE PARISH

A business meeting and social were held by the Lafayette Parish Auxiliary at the home of Mrs. R. D. Voorhies, with Mrs. J. O. Duhon as co-hostess. At this meeting Mrs. Edgar Breaux was appointed health education chairman.

ORLEANS PARISH

At the December meeting, Mrs. John T. Sanders was installed as President of the Orleans Parish Auxiliary succeeding Mrs. Boni J. DeLaureal.

Following are Officers and Executive Board for 1951:

President, Mrs. John T. Sanders; President-Elect, Mrs. Louis Leggio; 1st Vice-President, Mrs. Albert Prieto; 2nd Vice-President, Mrs. Edward Kagy; 3rd Vice-President, Mrs. Lloyd Kuhn; 4th Vice-President, Mrs. Howard Mahorner; Recording Secretary, Mrs. Abe Golden; Corresponding Secre-

tary, Mrs. Frank Marek; Treasurer, Mrs. Robert C. Kelleher; Secretary of Publicity, Mrs. Wm. Sodemann; Parliamentarian, Mrs. Monte Myers; Historian, Mrs. Wm. Rein.

Above Officers: Past-President, Mrs. Boni DeLaureal; Orleans Parish Medical Society President's Wife, Mrs. Boni DeLaureal; Chairmen of Standing Committees: Archives, Mrs. Wm. Rein; Commemoration Fund, Mrs. Lloyd Kuhn; Contact, Mrs. Louis Leggio; Conventions, Mrs. Joseph D'Antoni; Entertainment, Mrs. Eugene Countiss; Health Activities, Mrs. Harold Mahorner; Notifications, Mrs. R. C. Kelleher; Printing and Supplies, Mrs. Boni DeLaureal; Program, Mrs. Edward Kagy; Public Relations, Mrs. J. M. Davidson; Revisions, Mrs. Monte Myers; Social Activities, Mrs. Albert Prieto; Social Service Activities, Mrs. C. R. Walters.

Mrs. Edwin R. Guidry, contest chairman, announced that the Auxiliary will sponsor for the second year an essay contest on why the private practice of medicine furnishes this country with the finest medical care. The local contest is open to students in the city from the seventh grade through high school. The deadline is March 1 and essays are limited to 1500 words.

Mrs. Guidry is also contest chairman for the state auxiliary which is sponsoring the contest for the first time this year. Cash prizes are being awarded on local, state and national levels, she said. Last year a New Orleans student, Rose N. Agnew, won fourth place in the national contest.

Slogan for the Orleans Parish Auxiliary for 1951 is "Everyone manage to sponsor at least one essay."

At the January meeting of the Woman's Auxiliary to the Orleans Parish Medical Society Dr. Boni J. DeLaureal, newly installed President of the Society thanked the ladies for their cooperation in the past and asked their continued assistance to the Parish Society.

Subsequent to Dr. DeLaureal's talk Mrs. Ruth Moore Mathews gave a delightful dramatic reading.

Following precedent of several years two more outstanding members of the Auxiliary have been elected as honorary members. It is with a great deal of pleasure and pride that the group has bestowed this honor upon Mrs. Herman Gessner and Mrs. William Kohlman.

RAPIDES PARISH

In November a meeting of the Rapides Parish Auxiliary was held at the home of Mrs. Arthur L. Seale. Coffee hour was enjoyed after which a most interesting address on "The Menace of Socialism" was presented by Mr. Fred C. Barksdale.

WOMAN'S AUXILIARY TO THE LOUISIANA ACADEMY OF GENERAL PRACTICE

Members of the First and Second Districts of the Louisiana Woman's Auxiliary to the American Academy of General Practice met on the morning of November twenty-fifth. The occasion was a coffee party given by Mrs. Edwin Guidry at her home on Broadway. The featured event was the introduction of Mrs. George D. Feldner, State President, who gave an inspiring address concerning the good fellowship to be derived by members of the auxiliary who take active interest. Members who attended were: Mesdames John Atkinson, Vincent Blandino, George Feldner, Thomas Furlow, William Gillespie, Joel Gray, Edwin Guidry, P. P. LaBruyere, Bruno Mancuso, Joseph Massony, Daniel Murphy, Charles Robinson, M. J. St. Romain and F. Wild.

On December seventeenth, at the home of Dr. and Mrs. Daniel Murphy, members of the First and Second Districts Womans' Auxiliary to the Louisiana Academy of General Practice entertained their husbands at an old-fashioned Christmas Party. Numerous gifts were presented by Santa Claus himself and everyone took part in old-fashioned Christmas celebrations such as caroling, listening to "THE NIGHT BEFORE CHRISTMAS" and many other old but pleasant yuletide practices. Featured event of the night was the presentation of a gift from the Auxiliary to Dr. William Gillespie and his wife. The former was so honored because of his fifty years of medical service in the City of New Orleans.

Word from Mrs. A. J. Feder tells us that a district meeting of the Louisiana Academy of General Practice and their wives was held on January 11, at 3:30 p. m. at Cave Tangi in Hammond. Our state president and President-elect, and their husbands attended and Mrs. Feldner gave a fine address.

WOMAN'S AUXILIARY TO THE SOUTHERN MEDICAL ASSOCIATION

Following is report of meeting of the Woman's Auxiliary to the Southern Medical Association held in St. Louis, Mo., November 12-16, 1950:

The 26th Annual meeting of the Auxiliary opened with a breakfast for the Executive Board on November 14. This was followed by the first section of the general meeting at 10:00 A. M. One hundred and ninety-eight women were registered for the meeting and much interest was shown in Southern by the large attendance at the meetings and the social functions. Mrs. Robert C. Haynes, President, presided at all the sessions turning the gavel

over to Mrs. L. S. Thompson of Dallas, Texas in an impressive installation ceremony conducted by Mrs. F. F. Kirby of Waco, Texas.

Louisiana was very much in the limelight with two presidents on the program; Mrs. Herold, the National Auxiliary President was honored and feted during the entire meeting and Mrs. DeWitt T. Milam, our State President, gave a most interesting and humorous report of the A. M. A. meeting in San Francisco. She has been named Revisions Chairman of Southern for the coming year.

The first prize for Doctor's Day went to Louisiana, with the parishes of East Baton Rouge and Calcasieu both carrying the top honors; Baton Rouge for the issue of the local magazine "The Register" which was entirely dedicated to the doctors, and Calcasieu for the wonderful radio interview by the President of the Auxiliary with an Englishwoman now residing in Lake Charles on "Socialized Medicine in England."

Further honors have also come to your state with a New Orleans doctor and his wife designing the President's Pin for the Southern Auxiliary, which was fashioned after that of our Louisiana State Auxiliary pin.

Four hundred dollars were contributed to the Jane Todd Crawford Fund. This is the largest amount ever to be collected in one year. A dining-room set has been purchased by the Auxiliary, to furnish the room in honor of Jane Crawford in the Ephraim McDowell home in Kentucky. In the future, all money contributed to this fund will be used for the Post-Graduate Gynecology project.

Every Southern State observed Doctors' Day and every state used the official red carnation to honor their doctors.

Material from many of the states was contributed to the Romance and Research of Medicine file which is available to all members upon request.

Officers for 1950-51 are: President-Elect, Mrs. V. Eugene Holcombe, Charleston, West Va.; First Vice-President, Mrs. Neil Woodward, Oklahoma City, Okla.; Second Vice-President, Mrs. W. W. Crawford, Hattiesburg, Miss.; Recording Secretary, Mrs. Louis K. Hundley, Pine Bluff, Ark.; Corresponding Secretary, Mrs. Warren E. Massey, Dallas, Texas; Treasurer, Mrs. L. M. Jenkins, Miami, Fla.; Historian, Mrs. A. L. O'Briant, Raefor, N. C.; Parliamentarian, Mrs. Robert C. Haynes, Marshall, Missouri.

Mrs. George D. Feldner,
Louisiana Councilor
Southern Medical Auxiliary

BOOK REVIEWS

Cytologic Diagnosis of Lung Cancer: By Seymour M. Farber, M. D. & others. Springfield, Chas. C. Thomas, Publisher, 1950. Pp. 55, Col. pl. Price $6.00

Cystologic study of sputum and bronchial washings has become an important adjunct in the diagnosis of bronchogenic carcinoma. Dr. Farber and co-workers have had considerable experience in the study of cells from the lungs and have written a very clear, and concise volume aiding those who must interpret cells in the secretions. The colored photographs aid considerably those who are studying cells from pulmonary secretions and bring out various pitfalls of which one must be careful in making such a serious diagnosis. It is felt that this volume would be extremely valuable to those interested in earlier diagnosis of lung cancer, and subsequently, its earlier treatment.

JOSEPH ZISKIND, M. D.

The Cytologic Diagnosis of Cancer: By the staff of Vincent Memorial Laboratory & Vincent Memorial Hospital. Philadelphia, W. B. Saunders Co. 1950. pp. 229. Price $6.50.

Interest in cytologic diagnosis for malignancy is increasing steadily and much has been written on methods and interpretation of cells from various secretions of the body. This particular volume from the Vincent Memorial Laboratory is a very good one in aiding interpretations of various cellular elements for laboratory workers. It is well illustrated, and much fine detail is given which is so important for those not experienced with this type of diagnosis. This volume is highly recommended to those interested in cytologic study.

JOSEPH ZISKIND, M. D.

Techniques in British Surgery: By Rodney Maingot, 1950. W. B. Saunders Co., Philadelphia, London.

This volume consists of a series of monographs by 29 prominent surgeons. There is no pretense of completeness, the presentations being limited to a portrayal of the management of common surgical problems. The presentations are not limited to discussions and demonstrations of operative techniques, but include considerations of pathological anatomy, pathological physiology and both preoperative and postoperative treatment.

As stated in the preface, the book is typically British in format, illustration, and language. To illustrate the latter characteristic, in a discussion of the management of senile and diabetic gangrene, it is stated that "the use of a euphoriant is of great help in dispelling the atmosphere of inspissated gloom surrounding the patient".

The book is pitched more to the level of the experienced surgeon than to the level of medical stu-

dents and is, altogether, both readable and valuable as a reference work for clinical surgeons.

AMBROSE H. STORCK, M. D.

Bone and Joint Diseases: Pathology Correlated with Roentgenological and Clinical Features, by J. Vernon Luck, M. S., M. D., F. A. C. S. Springfield, Ill., Charles C. Thomas, Publisher, 1950. Pp. 614. Price, $16.50.

This is an excellent book which fills a long felt need for a single text on the pathology of bones and joints. The author first compiled this material as a set of lectures when he was associated with Dr. Aruthur Steindler at the University of Iowa over ten years ago. This correlation of clinical and roentgenological features with pathological anatomy is one of the best illustrated and most comprehensive this reviewer has had the pleasure of seeing.

Without a doubt this book will become a classic which belongs in the library of every surgeon interested in the treatment of pathological processes of the bones and joints, as well as pathologists who are asked to interpret pathological anatomy of the bones and joints.

JACK WICKSTROM, M. D.

The Clinical Use of Isotopes: By Bertram V. A. Low-Beer, M. D. Springfield, Ill., Charles C. Thomas, publisher, 1950. Pp. 436. Price, $9.50.

In a field as rapidly growing as that concerned with the use of radioisotopes, it is not possible for a review, such as this book is, to include all the latest developments. By the time the book was off the press, it was already about a year behind, particularly with reference to tracer studies.

The first section of the book, devoted to the task of acquainting the reader with the physical nature of the atom, radioactivity and instrumentation dealing with its detection and measurement, is adequate enough for those not concerned with the intricacies of nuclear physics but rather with a working knowledge of the atom as applied to biology. This section along with the appendices and section on dosage determination offers a valuable source for equations and tables applicable to radioactivity in biology.

The second section of the book, dealing with clinical investigation, is essentially a compendium of brief abstracts of the available literature, and in attempting to cover all the isotopes and all uses, much valuable detail is necessarily lost. There is no apparent evaluation of these abstracts, some of which contain misinterpretations of the data and present misleading conclusions to the reader. There are also erroneous statements of the physical characteristics of radioelements and their production.

In the last section of the book, the author concisely presents the uses and limitations of radioisotope therapy and appears to be on more familiar ground than when discussing varied physiology in the section on investigation.

Unfortunately errors in some of the references handicap one of the primary purposes of a book of this nature—its use as a bibliography.

Except for reference to the sections concerned with dosage determination of isotopes either for investigational or therapeutic purposes, the book offers no great advantage to the students for whom it is intended.

SAM A. THREEFOOT, M. D.

Doctor Come Quickly: By Frank J. Clancy, M. D. Seattle, Wash. Superior Pub. Co. 1950. Pp. 258. Price, $2.95.

Although drawn from the author's own professional experience, this volume is written essentially for and about patients rather than physicians. The author describes experiences as a hotel doctor, a general practitioner, and a specialist; the physician-patient relationship is stressed throughout. Physicians are urged from the days of medical school to treat the "patient as a person". *Doctor Come Quickly* suggests to the patient that the physician also should be treated "as a person", and turns the tables in advising the patient how to treat the doctor.

MARY LOUISE MARSHALL.

PUBLICATIONS RECEIVED

The Audiology Foundation, Chicago: The Audiology Clinic, by Moe Bergman, Ed. D.

The Blakiston Company, Philadelphia: Science of Health (2nd Edition), by Florence L. Meredith, B. Sc., M. D.

Federal Civil Defense Administration, Washington: Health Services and Special Weapons Defense, prepared by the Health Resource Office of the National Security Resources Board.

The C. V. Mosby Co., St. Louis: Methods in Medicine, by George R. Herrmann, M. D., Ph. D

W. B. Saunders Company, Philadelphia and London: Physical Diagnosis (4th Edition), by Ralph H. Major, M. D.; Functional Anatomy of the Limbs and Back, by W. Henry Hollinshead, A. B., M. S., Ph. D.

Charles C. Thomas, Publisher, Springfield, Ill.: The External Secretion of the Pancreas, by J. Earl Thomas, M. D.; Your Prostate Gland, by Reed M. Nesbit, M. D.

University of Kansas Press, Lawrence, Kansas: Recent Advances in Nutrition, by Paul R. Cannon and others.